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=> s (ACE(w)inhibitor or angiotensin(w)converting(w)enzyme(w)inhibitors)
L1       88349 (ACE(W) INHIBITOR OR ANGIOTENSIN(W) CONVERTING(W) ENZYME(W) INHI
               BITORS)

=> s l1 and (bone(w)morphogen or osteogenic(w)protein or bmp or op)
L2          66 L1 AND (BONE(W) MORPHOGEN OR OSTEOGENIC(W) PROTEIN OR BMP OR
               OP)

=> s l1 and proteinuria
L3        5814 L1 AND PROTEINURIA

=> s l2 and proteinuria
L4           6 L2 AND PROTEINURIA

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5           6 DUP REM L4 (0 DUPLICATES REMOVED)

=> dup rem l2
PROCESSING COMPLETED FOR L2
L6          48 DUP REM L2 (18 DUPLICATES REMOVED)

=> dis ibib abs l5 1-6

L5   ANSWER 1 OF 6  CAPLUS  COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER:        2009:1489395  CAPLUS
TITLE:                   Increased renoprotection with ACE
                         inhibitor plus aldosterone antagonist as
                         compared to monotherapies-the effect on podocytes
AUTHOR(S):               Nemeth, Zalan; Kokeny, Gabor; Godo, Maria; Mozes,
                         Miklos; Rosivall, Laszlo; Gross, Marie-Luise; Ritz,
                         Eberhard; Hamar, Peter
CORPORATE SOURCE:        Department of Pathophysiology, Semmelweis University,
                         Budapest, Hung.
SOURCE:                  Nephrology, Dialysis, Transplantation (2009), 24(12),
                         3640-3651
                         CODEN: NDTREA; ISSN: 0931-0509
PUBLISHER:               Oxford University Press
DOCUMENT TYPE:           Journal
LANGUAGE:                English
AB   Background.  Blockade of the renin-angiotensin-aldosterone system (RAAS)
     does not completely prevent progression of renal disease.
```

Mineralocorticoid receptor blockade provides addnl. renoprotection over ACE-inhibition monotherapy. We examined the mechanisms underlying superior renoprotection in the subtotal nephrectomy (SNX) model. Methods. Sprague-Dawley rats were randomized into six groups: (1) sham-op , (2) SNX without treatment, (3) SNX + quinapril (Q), (4) SNX + spironolactone (S), (5) SNX + combination therapy (Q+S), (6) SNX + combination hydrochlorothiazide + reserpin + hydralazine (HRH). Albuminuria and blood pressure were monitored, and kidneys were examined by morphometric and mol. methods. Results. In SNX rats, albumin excretion was significantly higher than in sham-op rats. Blood pressure reduction was not significantly different between the treatment groups. All therapies (S, Q, Q+S and HRH) reduced albuminuria; the values were lowest in animals treated with Q+S. The volume d. of glomerular matrix and the number of mesangial cells were significantly increased in SNX and were lowest in SNX treated with Q+S. The number of podocytes was reduced in SNX, but was normalized in SNX treated with Q+S. Glomerular vols. and podocyte vols. were significantly higher in SNX than in sham-op. Both vols. were reduced by all interventions, but almost normalized by treatment with Q+S. Expression of collagen IV, TGF-β1 and desmin was increased after SNX and significantly reduced by treatment with Q and Q+S. Conclusions. In subtotally nephrectomized rats, mineralocorticoid blockade provided addnl. renoprotection over and above ACE inhibition. Such benefit was paralleled by major changes in podocyte number and morphol. and was not blood pressure dependent.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 6 EMBASE

ACCESSION NUMBER: 2009536786 EMBASE

TITLE: [Changing the trandolapril dosage regimen does not affect the proteinuria-lowering effect of ACE inhibition in non-diabetic renal disease].
Wijziging doseerregime trandolapril heeft geen effect op proteinurieverlagend effect van ACE-remming bij niet-diabetische nierziekte.

AUTHOR: Van Der Wouden, E.A.; Henning, R.H.; De Zeeuw, D.
CORPORATE SOURCE: Klinische Farmacologie, Universitair Medisch Centrum Groningen. elsvdwouden@kpnplanet.nl
AUTHOR: Van Der Wouden, E.A.
CORPORATE SOURCE: UMC St Radboud, Nijmegen, Netherlands. elsvdwouden@kpnplanet.nl
AUTHOR: Vogt, L.; Navis, G.J.
CORPORATE SOURCE: Universitair Medisch Centrum Groningen.
AUTHOR: Vogt, L.
CORPORATE SOURCE: Academisch Medisch Centrum, Amsterdam, Netherlands.
AUTHOR: Hemmelder, M.H.
CORPORATE SOURCE: Medisch Centrum Leeuwarden.
AUTHOR: Van Der Wouden, E. A. (correspondence)
CORPORATE SOURCE: UMC St Radboud, Nijmegen, Netherlands. elsvdwouden@kpnplanet.nl

SOURCE: Pharmaceutisch Weekblad, (18 Sep 2009) Vol. 144, No. 38, pp. 156-159.
Refs: 10
ISSN: 0031-6911 CODEN: PHWEAW
PUBLISHER: Kon. Ned. Mij. ter Bevordering der Pharmacie (KNMP), P.O. Box 30460, The Hague, 2500 GL, Netherlands.
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 006 Internal Medicine
018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index

LANGUAGE: Dutch; Flemish
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 30 Nov 2009
Last Updated on STN: 30 Nov 2009

AB Objective: To investigate whether altering the dosing time of ACE inhibitors could overcome the relative nocturnal therapy resistance. ACE inhibitors are drugs of first choice to reduce proteinuria in renal diseases, but the antiproteinuric response may vary substantially. We previously observed a relative therapy resistance to blockade of the renin-angiotensin-aldosterone system (RAAS) during the night. Since higher residual proteinuria is associated with more rapid renal function loss, it is important to enhance the nocturnal antiproteinuric response. Design and methods: 14 non-diabetic proteinuric patients on stable RAAS inhibition, with residual proteinuria of >1 g/day were converted to trandolapril (4 mg) in the morning. Other antihypertensive medication was continued. After six weeks, patients were randomized to evening (4 mg) or twice daily dosing (2 mg) of trandolapril in a cross-over set-up (six weeks each). During the last study period patients again used trandolapril (4 mg) in the morning. Patients collected twice 24 h urine in daytime and nighttime portions every six-week period. Proteinuria and blood pressure were measured. Results: Total residual proteinuria and blood pressure were equal during all periods. Daytime and nighttime proteinuria were also comparable during all periods. Blood pressure day-night rhythm was similar during all periods. Evening dosing and twice daily dosing did not affect total residual proteinuria , daytime proteinuria or nighttime proteinuria.
Sodium and protein intake were not significantly different among the different dosing regimens. Conclusions: Altering the dosing time of the ACE inhibitor, trandolapril, does not increase the antiproteinuric response. Therefore, once daily dosing of the long-acting ACE inhibitor trandolapril at maximum dose results in its optimal antiproteinuric effect.

L5 ANSWER 3 OF 6 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
ACCESSION NUMBER: 2006:672161 BIOSIS
DOCUMENT NUMBER: PREV200600680607
TITLE: IgACE: First multicentric prospective double blind randomized and placebo controlled study on ACE-inhibitors (ACE-I) administration in moderately proteinuric IgA nephropathy (IgAN) in the young.
AUTHOR(S): Coppo, Rosanna; Peruzzi, Licia; Amore, Alessandro; Piccoli, Antonio; Cochat, Pierre; Stone, Rosario; Soergel, Marianne; Linne, Tommy; Project EEC Biomed BMH4 97 2487 [Reprint Author]
SOURCE: Nephrology Dialysis Transplantation, (JUL 2006) Vol. 21, No. Suppl. 4, pp. 293-294.
Meeting Info.: 43rd ERA-EDTA Congress. Glasgow, UK. July 15 -18, 2006. ERA; EDTA.
ISSN: 0931-0509.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Dec 2006
Last Updated on STN: 6 Dec 2006

L5 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2003:161291 CAPLUS
DOCUMENT NUMBER: 139:255033
TITLE: New approaches to delay the progression of chronic renal failure
AUTHOR(S): Klahr, Saulo; Morrissey, Jeremiah; Hruska, Keith;

Wang, Song; Chen, Qing
CORPORATE SOURCE: Departments of Internal Medicine, Cell Biology and Physiology, and Pediatrics, Washington University School of Medicine, St. Louis, MO, USA
SOURCE: Kidney International, Supplement (2002), 80, S23-S26
CODEN: KISUDF; ISSN: 0098-6577
PUBLISHER: Blackwell Science, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The mol. cloning of many bone morphogenetic proteins (BMP)-encoding genes and their identification as transforming growth factor-β (TGF-β) relatives enhanced the interest in these mols. and allowed expression and functional studies to be performed. Rats with ureteral obstruction were distributed into four groups. Group 1 received vehicle, group 2 received enalapril 12.5 mg/kg body wt/day, group 3 received 50 or 300 μg/kg body wt BMP-7, and group 4 received both enalapril and the high dose of BMP-7. We also studied the effects of BMP-7 administration in a model streptozocin-induced diabetes. Treatment with BMP-7 in rats with ureteral obstruction of 3 days duration and subsequent release indicated that this compound decreases interstitial volume and accelerates the return of renal function. After 16 wk of diabetes, the rats were treated with BMP-7. The administration of BMP-7 partially reversed renal hypertrophy, restored GFR to normal, and decreased proteinuria. These studies indicate that administration of BMP-7 maintains and restores renal function and structure in animals with ureteral obstruction and diabetic nephropathy.
REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 6 EMBASE
ACCESSION NUMBER: 2000417911 EMBASE
TITLE: [The protective role of ace inhibitors on the kidney function].
De beschermende rol van ACE-remmers op de nierfunctie.
AUTHOR: Lameire, N., Dr. (correspondence)
CORPORATE SOURCE: Nefrologie, Universitair Ziekenhuis, Ziekenhuis Gent, Gent, Belgium.
SOURCE: Tijdschrift voor Geneeskunde, (15 Nov 2000) Vol. 56, No. 22, pp. 1661-1662.
Refs: 7
ISSN: 0371-683X CODEN: TGEKBW
COUNTRY: Belgium
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 028 Urology and Nephrology
003 Endocrinology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
006 Internal Medicine
LANGUAGE: Dutch; Flemish
ENTRY DATE: Entered STN: 14 Dec 2000
Last Updated on STN: 14 Dec 2000

L5 ANSWER 6 OF 6 MEDLINE on STN
ACCESSION NUMBER: 1999164651 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10065257
TITLE: [Immunosuppressive treatment of patients with a nephrotic syndrome due to minimal change glomerulopathy].
Immunosuppressieve behandeling van patienten met een nefrotisch syndroom op basis van

'minimal-change'-glomerulopathie.
AUTHOR: Branten A J; Wetzels J F
CORPORATE SOURCE: Academisch Ziekenhuis, afd. Nierziekten, Nijmegen.
SOURCE: Nederlands tijdschrift voor geneeskunde, (1998 Dec 26) Vol. 142, No. 52, pp. 2832-8. Ref: 31
Journal code: 0400770. ISSN: 0028-2162. L-ISSN: 0028-2162.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: Dutch
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 26 Apr 1999
Last Updated on STN: 26 Apr 1999
Entered Medline: 13 Apr 1999
AB Prednisone monotherapy is the treatment of choice for patients with a nephrotic syndrome due to minimal change glomerulopathy. In adult patients treatment should be continued for at least 24 weeks. Within this period a remission of proteinuria will occur in 75 to 90% of the patients. Patients who do not respond satisfactorily to prednisone treatment can be treated with alkylating agents. Cyclophosphamide is the drug used most commonly. Prolonged treatment (> 12 weeks) is associated with a high risk of infertility. If alkylating agents cannot be used, prolonged treatment with ciclosporine is an option.

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FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 22:24:18 ON 05 MAY 2010
L1 88349 S (ACE(W)INHIBITOR OR ANGIOTENSIN(W)CONVERTING(W)ENZYME(W)INHIB
L2 66 S L1 AND (BONE(W)MORPHOGEN OR OSTEOGENIC(W)PROTEIN OR BMP OR OP
L3 5814 S L1 AND PROTEINURIA
L4 6 S L2 AND PROTEINURIA
L5 6 DUP REM L4 (0 DUPLICATES REMOVED)
L6 48 DUP REM L2 (18 DUPLICATES REMOVED)

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L6 ANSWER 1 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2010004089 EMBASE

TITLE: Review article: Anti-fibrotic agents for the treatment of Crohn's disease - Lessons learnt from other diseases.

AUTHOR: Szabo, H.; Fiorino, G.; Rovida, S.; Repici, A.; Malesci, A.C.; Danese, S. (correspondence)

CORPORATE SOURCE: Inflammatory Bowel Disease Unit, Division of Gastroenterology, Istituto Clinico Humanitas-IRCCS in Gastroenterology, Via Manzoni 56, 20089, Rozzano, Milan, Italy. sdanese@hotmail.com

AUTHOR: Fiorino, G.

CORPORATE SOURCE: GI Unit, Dipartimento di Scienze Cliniche, Policlinico Umberto i, Rome, Italy.

AUTHOR: Spinelli, A.

CORPORATE SOURCE: General Surgery III, University of Milan, Istituto Clinico Humanitas IRCCS, Rozzano, Milan, Italy.

AUTHOR: Malesci, A.C.

CORPORATE SOURCE: Department of Translational Medicine, University of Milan, Milan, Italy.

SOURCE: Alimentary Pharmacology and Therapeutics, (January 2010) Vol. 31, No. 2, pp. 189-201.
Refs: 125
ISSN: 0269-2813; E-ISSN: 1365-2036 CODEN: APTHEN

PUBLISHER: Blackwell Publishing Ltd, 9600 Garsington Road, Oxford, OX4 2XG, United Kingdom.

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; General Review; (Review)

FILE SEGMENT: 006 Internal Medicine
018 Cardiovascular Diseases and Cardiovascular Surgery
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
048 Gastroenterology

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 19 Jan 2010
Last Updated on STN: 19 Jan 2010

AB Background The current therapies for Crohn's disease (CD) are mainly focused on blockade of inflammation. Fibrosis remains one of the major complications of CD often leading to surgery, affecting patients' quality-of-life. Aim To summarize the published data regarding the potential anti-fibrotic role of drugs commonly used in CD and the most effective anti-fibrotic drugs used in other diseases evaluating their potential use to treat intestinal fibrosis in CD. Methods A literature search was performed in the PubMed, Medline, Cochrane and EMBASE databases, considering in vitro, animal and human studies on fibrosis in inflammatory bowel disease and other similar chronic pathologies. Results Treatment of fibrosis in CD is limited to surgery or endoscopic dilatation, although some of the drugs currently used may have anti-fibrotic activity. In other diseases, anti-fibrotic agents are already used or are in preclinical or clinical trials. ACE inhibitors, Angiotensin Receptor Blockers, and HMG-CoA inhibitors merit further investigation in CD because of their role in preventing fibrosis in cardiovascular and renal diseases. Conclusions Anti-fibrotic drugs are under evaluation or already used in clinical practice in other chronic inflammatory diseases. In CD, there is a great need for investigation into agents that may prevent, reduce or reverse intestinal fibrosis.

L6 ANSWER 2 OF 48 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2009779608 MEDLINE
DOCUMENT NUMBER: PubMed ID: 19666910
TITLE: Increased renoprotection with ACE inhibitor plus aldosterone antagonist as compared to monotherapies--the effect on podocytes.
AUTHOR: Nemeth Zalan; Kokeny Gabor; Godo Maria; Mozes Miklos; Rosivall Laszlo; Gross Marie-Luise; Ritz Eberhard; Hamar Peter
CORPORATE SOURCE: Department of Pathophysiology, Semmelweis University, Budapest, Hungary.
SOURCE: Nephrology, dialysis, transplantation : official publication of the European Dialysis and Transplant Association - European Renal Association, (2009 Dec) Vol. 24, No. 12, pp. 3640-51. Electronic Publication: 2009-08-08.
Journal code: 8706402. E-ISSN: 1460-2385. L-ISSN: 0931-0509.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 201002
ENTRY DATE: Entered STN: 28 Nov 2009
Last Updated on STN: 3 Feb 2010
Entered Medline: 2 Feb 2010
AB BACKGROUND: Blockade of the renin-angiotensin-aldosterone system (RAAS) does not completely prevent progression of renal disease. Mineralocorticoid receptor blockade provides additional renoprotection over ACE-inhibition monotherapy. We examined the mechanisms underlying superior renoprotection in the subtotal nephrectomy (SNX) model. METHODS: Sprague-Dawley rats were randomized into six groups: (1) sham-op , (2) SNX without treatment, (3) SNX + quinapril (Q), (4) SNX + spironolactone (S), (5) SNX + combination therapy (Q+S), (6) SNX + combination hydrochlorothiazide + reserpin + hydralazine (HRH). Albuminuria and blood pressure were monitored, and kidneys were examined by morphometric and molecular methods. RESULTS: In SNX rats, albumin excretion was significantly higher than in sham-op rats. Blood pressure reduction was not significantly different between the treatment groups. All therapies (S, Q, Q+S and HRH) reduced albuminuria; the values were lowest in animals treated with Q+S. The volume density of glomerular matrix and the number of mesangial cells were significantly increased in SNX and were lowest in SNX treated with Q+S. The number of podocytes was reduced in SNX, but was normalized in SNX treated with Q+S. Glomerular volumes and podocyte volumes were significantly higher in SNX than in sham-op. Both volumes were reduced by all interventions, but almost normalized by treatment with Q+S. Expression of collagen IV, TGF-beta(1) and desmin was increased after SNX and significantly reduced by treatment with Q and Q+S. CONCLUSIONS: In subtotally nephrectomized rats, mineralocorticoid blockade provided additional renoprotection over and above ACE inhibition. Such benefit was paralleled by major changes in podocyte number and morphology and was not blood pressure dependent.

L6 ANSWER 3 OF 48 EMBASE
ACCESSION NUMBER: 2009154669 EMBASE
TITLE: [Increased risk of complex regional pain syndrome with ACE-inhibitors].
Toegenomen risico op complex regionaal pijnsyndroom bij ACE-remmers.

AUTHOR: Smulders, Yvo
SOURCE: Nederlands Tijdschrift voor Geneeskunde, (21 Mar 2009) Vol. 153, No. 12, pp. 567.
Refs: 1
ISSN: 0028-2162 CODEN: NETJAN
PUBLISHER: Bohn Stafleu Van Loghum bv, P.O. Box 246, Houten, 3990 GA, Netherlands.
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
029 Clinical and Experimental Biochemistry
037 Drug Literature Index
LANGUAGE: Dutch; Flemish
ENTRY DATE: Entered STN: 15 Apr 2009
Last Updated on STN: 15 Apr 2009

L6 ANSWER 4 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2009536786 EMBASE
TITLE: [Changing the trandolapril dosage regimen does not affect the proteinuria-lowering effect of ACE inhibition in non-diabetic renal disease].
Wijziging doseerregime trandolapril heeft geen effect op proteinurieverlagend effect van ACE-remming bij niet-diabetische nierziekte.
AUTHOR: Van Der Wouden, E.A.; Henning, R.H.; De Zeeuw, D.
CORPORATE SOURCE: Klinische Farmacologie, Universitair Medisch Centrum Groningen. elsvdwouden@kpnplanet.nl
AUTHOR: Van Der Wouden, E.A.
CORPORATE SOURCE: UMC St Radboud, Nijmegen, Netherlands. elsvdwouden@kpnplanet.nl
AUTHOR: Vogt, L.; Navis, G.J.
CORPORATE SOURCE: Universitair Medisch Centrum Groningen.
AUTHOR: Vogt, L.
CORPORATE SOURCE: Academisch Medisch Centrum, Amsterdam, Netherlands.
AUTHOR: Hemmelder, M.H.
CORPORATE SOURCE: Medisch Centrum Leeuwarden.
AUTHOR: Van Der Wouden, E. A. (correspondence)
CORPORATE SOURCE: UMC St Radboud, Nijmegen, Netherlands. elsvdwouden@kpnplanet.nl
SOURCE: Pharmaceutisch Weekblad, (18 Sep 2009) Vol. 144, No. 38, pp. 156-159.
Refs: 10
ISSN: 0031-6911 CODEN: PHWEAW
PUBLISHER: Kon. Ned. Mij. ter Bevordering der Pharmacie (KNMP), P.O. Box 30460, The Hague, 2500 GL, Netherlands.
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 006 Internal Medicine
018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
LANGUAGE: Dutch; Flemish
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 30 Nov 2009
Last Updated on STN: 30 Nov 2009
AB Objective: To investigate whether altering the dosing time of ACE inhibitors could overcome the relative nocturnal therapy resistance. ACE inhibitors are drugs of first choice to reduce proteinuria in renal diseases, but the antiproteinuric response may vary substantially. We previously observed a relative therapy resistance to blockade of the renin-angiotensin-aldosterone system (RAAS) during the night. Since higher residual proteinuria is associated with

more rapid renal function loss, it is important to enhance the nocturnal antiproteinuric response. Design and methods: 14 non-diabetic proteinuric patients on stable RAAS inhibition, with residual proteinuria of >1 g/day were converted to trandolapril (4 mg) in the morning. Other antihypertensive medication was continued. After six weeks, patients were randomized to evening (4 mg) or twice daily dosing (2 mg) of trandolapril in a cross-over set-up (six weeks each). During the last study period patients again used trandolapril (4 mg) in the morning. Patients collected twice 24 h urine in daytime and nighttime portions every six-week period. Proteinuria and blood pressure were measured. Results: Total residual proteinuria and blood pressure were equal during all periods. Daytime and nighttime proteinuria were also comparable during all periods. Blood pressure day-night rhythm was similar during all periods. Evening dosing and twice daily dosing did not affect total residual proteinuria, daytime proteinuria or nighttime proteinuria. Sodium and protein intake were not significantly different among the different dosing regimens. Conclusions: Altering the dosing time of the ACE inhibitor, trandolapril, does not increase the antiproteinuric response. Therefore, once daily dosing of the long-acting ACE inhibitor trandolapril at maximum dose results in its optimal antiproteinuric effect.

L6 ANSWER 5 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
ACCESSION NUMBER: 2009:146545 BIOSIS
DOCUMENT NUMBER: PREV200900146545
TITLE: Anomalous origin of the left coronary artery from the pulmonary artery (alcapa) in adolescent: Presentation of clinical case.
AUTHOR(S): Rengel, J.; Figueira, J.; Galindo, A.; Sanchez, S.; Donis, I.; Figueredo, J.; Gonzalez, J.; Guerra, F.; Acosta, Y.; Tolj, I.
SOURCE: Circulation, (SEP 16 2008) Vol. 118, No. 12, pp. E220. Meeting Info.: World Congress of Cardiology. Buenos Aires, ARGENTINA. May 18 -21, 2008. World Heart Federat; Argentine Soc Cardiol; Argentine Federat Cardiol.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 25 Feb 2009
Last Updated on STN: 25 Feb 2009

AB Anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA) is a rare congenital anomaly that is one of the most common causes of myocardial ischemia and infarction in children; it results in a mortality rate of up 90% within the first year of life if it was not treated. It represents 0.5% of all congenital heart defects. 12 years old adolescent, was diagnosis with an acyanotic congenital heart defect (ACHD); type severe mitral insufficiency, on treatment with diuretics and digitalis. When she was 10 years old, cardiac catheterization demonstrated anomaly of origin the left coronary artery, arising from trunk of the pulmonary artery. She was referred to our center for resolution. Electrocardiogram: Signs of left ventricular hypertrophy. Echocardiography with color-flow: Coronaries dilated: right of 7.7mm, left of 8.3mm, no evidence of the origin of the left coronary artery, aorta tri-valve. Left cavities dilated, sigmoidal valves were competent, mitral valve thickened with elongation of septal valve and prolepses of both valves when closing, turbulent flow of insufficiency (regurgitation) that extends by the sidewall of the left atrium to the top and flows back into the pulmonary veins causing dilatation. Systolic left and right ventricular functions were conserved. After median sternotomy and pericardiotomy we found the typical location of the anomalous left coronary artery from posterior sinus of the main pulmonary artery, which

was dissected and reimplanted in the lateral face of the aorta. The sinus was reconstructed with bovine pericardial patch. The mitral valve was inspected then and decided to put a DURAN ring 29mm. Cardiopulmonary bypass time was 162 minutes and 131 minutes of Aortic cross clamp. Post op in ICU was satisfactory, ex-tubated after 24 hours and transferred to hospitalization at 72 hours, treatment with diuretics, ACE inhibitors and salicylic acetyl acid. To 4(a) day of post op presents signs of pericardial effusion that improve with anti-inflammatory non steroids and steroids. Echocardiogram control a week after surgery: moderate pericardial effusion without hemodynamic repercussion, maintaining same therapeutic conduct. Echocardiogram 2 weeks after: Enlarged coronary: right of 9.3mm dilated; left of 9.5mm with adequate flow. Mitral valve with Duran ring without prolapsed of the valves in their closing, slight turbulent flow, eccentric insufficiency extending by the sidewall of the left atrium with adequate systolic ventricular function. To the 6 months of PO, good general conditions with suitable physical activity for its age.

L6 ANSWER 6 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2007:351908 CAPLUS
DOCUMENT NUMBER: 146:373015
TITLE: TDF-related compounds and analogs thereof for detecting, preventing, and treating TDF-associated disorders
INVENTOR(S): Carlson, William D.; Keck, Peter C.; Sworin, Michael; Bosukonda, Dattatreymurty
PATENT ASSIGNEE(S): Thrasos Therapeutics, Inc., USA
SOURCE: PCT Int. Appl., 292pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2007035872	A2	20070329	WO 2006-US36830		20060920
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2006292147	A1	20070329	AU 2006-292147		20060920
CA 2623415	A1	20070329	CA 2006-2623415		20060920
EP 1945258	A2	20080723	EP 2006-815103		20060920
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, RS					
JP 2009508532	T	20090305	JP 2008-532384		20060920
PRIORITY APPLN. INFO.:			US 2005-719014P	P	20050920
			WO 2006-US36830	W	20060920

OTHER SOURCE(S): MARPAT 146:373015
AB The present invention relates generally to tissue differentiation factor (TDF) analogs. More specifically, the invention relates to structure-based methods and compns. useful in designing, identifying, and

producing mols., which act as functional modulators of TDF-like receptors. The invention further relates to methods of detecting, preventing, and treating TDF-associated disorders.

L6 ANSWER 7 OF 48 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2007484811 MEDLINE
DOCUMENT NUMBER: PubMed ID: 17703430
TITLE: The blockade of the renin-angiotensin system reverses tacrolimus related cardiovascular toxicity at the histopathological level.
AUTHOR: Agirbasli Mehmet; Papila-Topal Nurdan; Ogutmen Betul; Deniz Hicran; Cakalagaoglu Fulya; Tuglular Serhan; Akoglu Emel
CORPORATE SOURCE: Department of Cardiology, Marmara University Medical School, Istanbul, Turkey.. agirbasli@gmail.com
SOURCE: Journal of the renin-angiotensin-aldosterone system : JRAAS, (2007 Jun) Vol. 8, No. 2, pp. 54-8.
Journal code: 100971636. ISSN: 1470-3203. L-ISSN: 1470-3203.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200709
ENTRY DATE: Entered STN: 18 Aug 2007
Last Updated on STN: 27 Sep 2007
Entered Medline: 26 Sep 2007
AB INTRODUCTION: In this study, we investigate the toxic effects of tacrolimus (FK506) on the cardiovascular system at the histopathological level in a rat model and whether these effects can be reversed by the blockade of the renin-angiotensin system (RAS) by either an angiotensin-converting enzyme inhibitor (ACE-inhibitors) or an angiotensin receptor antagonist (ARB). METHODS AND RESULTS: Thirty-one Wistar rats were divided into four groups. FK506 group was treated with FK506 intraperitoneally (i.p.), FK506+ACE-inhibitors and FK506+ARB groups were treated with either quinapril or valsartan orally in addition to FK506. Control group was treated with saline i.p. Histological and immunohistochemical staining of cardiovascular tissue in the FK506 group showed increased vacuolar degeneration (11.2 vs. 5.8, p=0.008), arterial hyalinosis (10.7 vs. 6.3, p=0.036), transforming growth factor-beta (TGF-beta) (12.2 vs. 4.8, p=0.001) and vascular endothelial growth factor expression (VEGF) (10.7 vs. 6.3, p=0.036), elastic van Gieson (11.5 vs. 5.5, p=0.004), and periodic acid Schiff stain scores (12.5 vs. 4.5, p<0.001) compared to the control group. Immunohistochemical scores showed that expression of TGF-beta is up-regulated, and bone morphogenic protein (BMP-7) is down-regulated with FK506 toxicity. Adding RAS blockade with either an ACE-inhibitor or an ARB could reverse FK506 induced changes. Both FK506+ACE-inhibitors and FK506+ARB groups demonstrated decrease in arterial hyalinosis (22.1 vs. 14.4 (FK506+ACE-inhibitor) and 13.6 (FK506+ARB), p=0.09) and vacuolar degeneration (23.1 vs. 16.1 (FK506+ACE-inhibitor) and 12.4 (FK506+ARB), p=0.006) scores compared to the FK506 group. CONCLUSION: Blockade of RAS could reverse the histopathological signs of FK506 induced cardiac toxicity in a rat model.

L6 ANSWER 8 OF 48 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2006627901 MEDLINE
DOCUMENT NUMBER: PubMed ID: 17061436
TITLE: [The use of angiotensin converting enzyme (ACE) inhibitors during pregnancy clearly increases the risk of congenital malformations].
Duidelijk verhoogd risico op congenitale

afwijkingen door het gebruik van angiotensineconverterend-enzym(ACE)-remmers in de zwangerschap.
AUTHOR: de Jong-van den Berg L T W; Bakker M K; de Walle H E K; van den Berg P B
SOURCE: Nederlands tijdschrift voor geneeskunde, (2006 Oct 7) Vol. 150, No. 40, pp. 2222-3.
Journal code: 0400770. ISSN: 0028-2162. L-ISSN: 0028-2162.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Commentary
Letter
LANGUAGE: Dutch
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200611
ENTRY DATE: Entered STN: 26 Oct 2006
Last Updated on STN: 19 Dec 2006
Entered Medline: 30 Nov 2006

L6 ANSWER 9 OF 48 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2006385856 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16741184
TITLE: Inflammation and atherosclerosis: novel insights into plaque formation and destabilization.
AUTHOR: Stoll Guido; Bendszus Martin
CORPORATE SOURCE: Department of Neurology, Julius-Maximilians-Universitat, Wurzburg, Germany.. stoll_g@klinik.uni-wuerzburg.de
SOURCE: Stroke; a journal of cerebral circulation, (2006 Jul) Vol. 37, No. 7, pp. 1923-32. Electronic Publication: 2006-06-01. Ref: 136
Journal code: 0235266. E-ISSN: 1524-4628. L-ISSN: 0039-2499.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200607
ENTRY DATE: Entered STN: 28 Jun 2006
Last Updated on STN: 19 Jul 2006
Entered Medline: 18 Jul 2006
AB BACKGROUND AND PURPOSE: The simplistic view of atherosclerosis as a disorder of pathological lipid deposition has been redefined by the more complex concept of an ongoing inflammatory response. SUMMARY OF REVIEW: Apolipoprotein E and low-density lipoprotein (LDL)-receptor-deficient mice develop accelerated atherosclerosis allowing in-depth pathophysiological investigations. Atherosclerotic plaques in these mice contain large numbers of T cells and macrophages. Crossbreeding apolipoprotein E-deficient mice with T-cell-deficient mice and mice with impaired macrophage function (osteopetrotic op/op mice) disclosed the important impact of immune cells on atherosclerotic lesion development. In contrast to the detrimental role of T cells and macrophages, B cells appear to be atheroprotective. These basic experimental findings have partly been confirmed in studies of the human carotid artery system. Inflammation is not only instrumental in the development of human atheromatous plaques, but, importantly, plays a crucial role in the destabilization of internal carotid artery plaques, thus converting chronic atherosclerosis into an acute thrombo-embolic disorder. Humoral factors involved in internal carotid artery destabilization include cytokines, cyclooxygenase-2, matrix metalloproteinases, and tissue factor. Antibodies to oxidized LDL can reflect disease activity on one hand, but can also confer

atheroprotection. Novel MRI techniques may aid in the in vivo assessment of acute plaque inflammation in humans. CONCLUSIONS: The impact of inflammation on the development of atherosclerotic plaques and their destabilization opens new avenues for treatment. The effects of statins, acetylsalicyclic acid and angiotensin-converting enzyme inhibitors on stroke prevention may partly be attributable to their profound anti-inflammatory actions. Vaccination against modified LDL and heat shock proteins halt plaque progression in experimental atherosclerosis. Their potential for prevention of human atherosclerosis is currently under investigation.

L6 ANSWER 10 OF 48 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 2006477255 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16901062
TITLE: [The use of angiotensin converting enzyme (ACE) inhibitors during pregnancy clearly increases the risk of congenital malformations].
Duidelijk verhoogd risico op congenitale afwijkingen door het gebruik van angiotensineconverterend-enzym(ACE)-remmers in de zwangerschap.
AUTHOR: de Leeuw P W
CORPORATE SOURCE: Academisch Ziekenhuis Maastricht, afd. Interne Geneeskunde, Postbus 5800, 6202 AZ Maastricht..
p.deleeuw@intmed.unimaas.nl
SOURCE: Nederlands tijdschrift voor geneeskunde, (2006 Jul 22) Vol. 150, No. 29, pp. 1605-7.
Journal code: 0400770. ISSN: 0028-2162. L-ISSN: 0028-2162.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Dutch
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200608
ENTRY DATE: Entered STN: 12 Aug 2006
Last Updated on STN: 1 Sep 2006
Entered Medline: 31 Aug 2006
AB Recently, an important study was published concerning the possible teratogenic effects of angiotensin converting enzyme (ACE) inhibitors. Non-diabetic women who had used an ACE inhibitor during the first trimester of pregnancy had a significantly greater chance of giving birth to an infant with congenital malformations than women who had used no or other antihypertensive agents. The excess risk remained even after adjusting for several potential confounders. The results indicate that ACE inhibitors should not be prescribed to women who are likely to become pregnant during the course of treatment.

=> dis ibib abs l6 11-20
YOU HAVE REQUESTED DATA FROM FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' - CONTINUE? (Y)/N:y

L6 ANSWER 11 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2006:262914 CAPLUS
DOCUMENT NUMBER: 145:284454
TITLE: Inhibition of the Renin Angiotensin System Decreases Fibrogenic Cytokine Expression in Tacrolimus Nephrotoxicity in Rats

AUTHOR(S): Deniz, H.; Oeguetmen, B.; Cakalagaoglu, F.; Tuglular, S.; Oezener, C.; Akoglu, E.
CORPORATE SOURCE: Department of Internal Medicine, Marmara University Medical School, Istanbul, Turk.
SOURCE: Transplantation Proceedings (2006), 38(2), 483-486
CODEN: TRPPA8; ISSN: 0041-1345
PUBLISHER: Elsevier Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The aim of our study was to investigate the influence of angiotensin-converting enzyme (ACE) inhibition and angiotensin II receptor blockage on the renal function by light microscopic and immunohistochem. findings in a rat model of tacrolimus nephrotoxicity. Thirty-two male Wistar rats were divided into four groups of eight: G1 = control group; G2-G3, G4 = Tacrolimus (Tac) 1 mg/kg/d i.p. (i.p.); G3 (Tac + Q) = i.p. Tac and peroral quinapril 10 mg/kg; and G4 (Tac + V) = Tac and valsartan 40 mg/d. Serum blood urea nitrogen (BUN), creatinine, and creatinine clearance were measured before and at the end of the study period. Renal tissues were assessed for light microscopic findings of tacrolimus toxicity. Transforming growth factor-β, VEGF, PDGF, BMP-7, and interleukin-6 (IL-6) expression were semiquant. scored after immunohistochem. staining. At the end of the study period serum BUN and creatinine levels were increased in all groups, but creatinine clearance was not significantly changed between the groups. Afferent arteriolopathy was significantly less pronounced in G3 vs. G2 and G4. Interstial fibrosis was significantly less pronounced in G3 and G4 vs. G2. TGF-β, PDGF, and IL-6 expression were significantly increased in G2, G3, and G4 compared to G1, and in G2 compared to G3 and G4. BMP -7 expression was significantly decreased in G2, G3, and G4 compared to G1, whereas the differences between G2, G3, and G4 failed to reach statistical significance. In conclusion, the results of our study suggested that renin angiotensin inhibition down-regulates fibrogenic cytokine expression in rats displaying tacrolimus nephrotoxicity.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 12 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
ACCESSION NUMBER: 2006:672161 BIOSIS
DOCUMENT NUMBER: PREV200600680607
TITLE: IgACE: First multicentric prospective double blind randomized and placebo controlled study on ACE-inhibitors (ACE-I) administration in moderately proteinuric IgA nephropathy (IgAN) in the young.
AUTHOR(S): Coppo, Rosanna; Peruzzi, Licia; Amore, Alessandro; Piccoli, Antonio; Cochat, Pierre; Stone, Rosario; Soergel, Marianne; Linne, Tommy; Project EEC Biomed BMH4 97 2487 [Reprint Author]
SOURCE: Nephrology Dialysis Transplantation, (JUL 2006) Vol. 21, No. Suppl. 4, pp. 293-294.
Meeting Info.: 43rd ERA-EDTA Congress. Glasgow, UK. July 15 -18, 2006. ERA; EDTA.
ISSN: 0931-0509.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Dec 2006
Last Updated on STN: 6 Dec 2006

L6 ANSWER 13 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights

reserved on STN
ACCESSION NUMBER: 2005562369 EMBASE
TITLE: Prevention of nephropathy in patients with type 2 diabetes mellitus.
AUTHOR: Tzamaloukas, Antonios H. (correspondence); Murata, Glen H.
CORPORATE SOURCE: Sections of Nephrology and General Internal Medicine, New Mexico Veterans Affairs Health Care System and Department of Medicine, University of New Mexico School of Medicine, 1501 San Pedro, SE, Albuquerque, NM 87108, United States. Antonios.tzamaloukas@med.va.gov
SOURCE: International Urology and Nephrology, (Sep 2005) Vol. 37, No. 3, pp. 655-663.
Refs: 40
ISSN: 0301-1623 CODEN: IURNAE
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 028 Urology and Nephrology
003 Endocrinology
037 Drug Literature Index
006 Internal Medicine
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 29 Dec 2005
Last Updated on STN: 29 Dec 2005

AB The rising incidence of type 2 diabetes mellitus and of its complications will make it the most important health care challenge in the first quarter of the 21st Century. Diabetic nephropathy left unchecked will overwhelm the renal resources. Simple methods (proper diet and exercise, prevention of obesity) are successful in preventing type 2 diabetes in the great majority of the persons at risk. In patients with established type 2 diabetes, nephropathy can be prevented or greatly delayed by strict metabolic control, strict control of blood pressure using angiotensin-converting enzyme
inhibitors and angiotensin receptor blockers as the first line of drugs, tight control of serum lipids using statins as indicated, low protein diet, avoidance of smoking and other nephrotoxic influences, prevention of abnormalities in calcium/phosphorus metabolism, and prevention of renal anemia by the early use of erythropoietin. Current research offers the promise of definitive prevention of both type 2 diabetes and diabetic nephropathy. .COPYRGT. Springer 2005.

L6 ANSWER 14 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2004:203626 CAPLUS
DOCUMENT NUMBER: 140:247061
TITLE: Conjoint administration of morphogens and ACE inhibitors in treatment of chronic renal failure
INVENTOR(S): Charette, Marc F.; Hruska, Keith A.; McCartney, John
PATENT ASSIGNEE(S): Curis, Inc., USA; Washington University in St. Louis; Barnes-Jewish Hospital
SOURCE: PCT Int. Appl., 295 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004019876	A2	20040311	WO 2003-US26923	20030828
WO 2004019876	A3	20060323		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,

CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2497048	A1	20040311	CA 2003-2497048		20030828
AU 2003268219	A1	20040319	AU 2003-268219		20030828
AU 2003268219	B2	20090924			
EP 1578360	A2	20050928	EP 2003-749170		20030828
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK					
US 20050272649	A1	20051208	US 2003-650326		20030828
JP 2006516020	T	20060615	JP 2004-531612		20030828
AU 2009250981	A1	20100114	AU 2009-250981		20091216
PRIORITY APPLN. INFO.:			US 2002-406431P	P	20020828
			AU 2003-268219	A3	20030828
			WO 2003-US26923	W	20030828

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The present invention provides reagents and methods for the treatment, and pharmaceuticals for use in the prevention and/or treatment, of chronic renal failure and other renal disorders in subjects (particularly mammalian subjects) renal replacement therapy. The methods involve the conjoint administration of ACE (Angiotensin-Converting Enzyme) inhibitors or Angiotensin II Receptor Antagonists (AIIRAs) with one or more OP/BMP family of proteins (morphogens, or inducers of morphogens, or agonists of the corresponding morphogen receptors, etc.). The invention also provides methods for implantation of renal cells induced with the conjoint administration of ACE inhibitors or AIIRAs with those morphogens.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

L6 ANSWER 15 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

ACCESSION NUMBER: 2005:527033 BIOSIS
DOCUMENT NUMBER: PREV200510316996
TITLE: Targeted use of amiodarone to reduce atrial arrhythmias after cardiac surgery: The prophylactic amiodarone for the prevention of arrhythmias that begin early after revascularization, valve repair, or replacement (PAPABEAR) trial.
AUTHOR(S): Mitchell, L. B. [Reprint Author]; Wyse, D. G.; Connolly, Carol J.; Prystai, Gregory D.; Bayes, Alexander J.; Kidd, William T.; Kieser, Teresa M.; Burgess, John J.; Ferland, Andre; MacAdams, Charles L.; Maitland, Andrew; Exner, Derek V.
CORPORATE SOURCE: Libin Cardiovasc Inst Alberta, Calgary, AB, Canada
SOURCE: Circulation, (OCT 26 2004) Vol. 110, No. 17, Suppl. S, pp. 741.
Meeting Info.: 77th Scientific Meeting of the American-Heart-Association. New Orleans, LA, USA. November 07 -10, 2004. Amer Heart Assoc.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Dec 2005

Last Updated on STN: 1 Dec 2005

AB Background: Atrial tachyarrhythmias (AT) are common complications of cardiac surgery and increase morbidity, hospital stay, and costs. The PAPABEAR trial was a randomized comparison of amiodarone (N=299) vs. placebo (N=301) for prevention of AT after cardiac surgery. Overall, amiodarone therapy reduced the incidence of post-operative AT (hazard ratio [HR] 0,48; p<0.001). The absolute risk reduction (ARR) was 14%. Thus, the number of patients needed to treat (NNT) with amiodarone to prevent one from developing post-operative AT was 7.1. The present analysis tests the hypothesis that baseline characteristics can identify patients most likely to benefit from targeted use of prophylactic amiodarone. Methods. Common baseline characteristics (those present in >= 10% of the population) were identified and their capacity to identify patients at higher risk of AT were evaluated using univariate and multivariate (forward stepwise) Cox models. Results: Baseline characteristics evaluated included age, gender, heart rate, blood pressure, diabetes, heart failure, hypertension, NYHA class, medications (beta-blocker, ACE inhibitor, digoxin, anticoagulants, aspirin, NSAIDS), and type of surgery. In both univariate and multivariate models age :65 years (univariate HR 2.0; p<0.001; multivariate HR 1.9; p=0.001) and valve vs. non-valve surgery (univariate HR 2.0; p<0.001; multivariate HR 1.9; p=0.02) predicted post-op AT. Beta-blocker use was associated with a trend toward a lower AT risk on univariate analysis (HR 0.7; p=0.07), but not on multivariate analysis (HR 0,9; p=0.8). The 22 patients <65 years of age not having valve surgery benefited from amiodarone (HR 0.4; p=0.03), but the ARR was small (10%), and the NNT large (9.8). Similar results were observed for the 284 pts with one but not both risk factors (HR 0.5; p=0.01; ARR 13%; NNT 7.8). The 93 pts with both factors had a similar relative benefit HR 0.4; p=0.007), a larger ARR (29%), and a smaller NNT (3.5). Conclusions: Age >= 65 years and valve surgery identify patients most likely to benefit from prophylactic amiodarone therapy. Treating 100 patients with neither characteristic will prevent 10 from having post-operative AT, while treating 100 patients with both characteristics will prevent 29 from having AT.

L6 ANSWER 16 OF 48 EMBASE

ACCESSION NUMBER: 2004199134 EMBASE
TITLE: Mechanisms of tubulointerstitial fibrosis.
AUTHOR: Iwano, Masayuki
CORPORATE SOURCE: First Dept. of Internal Medicine, Nara Medical University, Kashihara, Nara, Japan.
AUTHOR: Neilson, Eric G., Dr. (correspondence)
CORPORATE SOURCE: Depts. Med. Cell and Devmtl. Biol., Vanderbilt University, School of Medicine, Nashville, TN, United States. eric.neilson@vanderbilt.edu
AUTHOR: Neilson, Eric G., Dr. (correspondence)
CORPORATE SOURCE: Department of Medicine, D-3100 MCN, Vanderbilt Univ. School of Medicine, Nashville, TN 37232-2358, United States. eric.neilson@vanderbilt.edu
SOURCE: Current Opinion in Nephrology and Hypertension, (May 2004) Vol. 13, No. 3, pp. 279-284.
Refs: 50
ISSN: 1062-4821 CODEN: CNHYEM
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 028 Urology and Nephrology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 17 Jun 2004

Last Updated on STN: 17 Jun 2004

AB Purpose of review: Tubulointerstitial fibrosis is the final common pathway to endstage renal disease. Understanding the mechanisms of tubulointerstitial fibrosis is essential in establishing novel therapeutic strategies for the prevention or arrest of progressive kidney diseases. The present review focuses on a newly proposed mechanism of tubulointerstitial fibrosis, one that emphasizes the roles of epithelial-mesenchymal transition and cellular activation. Recent findings: Among the cells that accumulate in the renal interstitium, fibroblasts are the principal effectors mediating tubulointerstitial fibrosis. By contrast, the phagocytosis of extracellular matrix and apoptotic cells by macrophages may actually exert a beneficial effect. Interstitial fibroblasts are more heterogeneous than expected, and during renal fibrosis new fibroblasts are derived mainly through epithelial-mesenchymal transition. The intracellular signaling pathways leading to initiation of epithelial-mesenchymal transition remain largely unknown, though recent studies have identified β-catenin and Smad3 activation of lymphoid enhancer factor, integrin-linked kinase, and small GTPases and mitogen-activated protein kinases as key components. Transforming growth factor-β is believed to be a critical fibrogenic factor, but recent studies have also focused on transforming growth factor-β independent pathways as mechanisms of tubulointerstitial fibrosis. As the mechanisms underlying tubulointerstitial fibrosis leading to epithelial-mesenchymal transition have been identified, so have cytokines that efficiently antagonize renal fibrosis, particularly bone morphogenic protein-7 and hepatocyte growth factor. Summary: In combination with traditional angiotensin converting enzyme inhibitors, newly identified cytokines may eventually form the basis for new therapeutic strategies aimed at inhibiting the progression of renal disease. .COPYRGT. 2004 Lippincott Williams & Wilkins.

L6 ANSWER 17 OF 48 MEDLINE on STN DUPLICATE 6

ACCESSION NUMBER: 2004123861 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15013323
TITLE: Cyclosporine-A induced nephrotoxicity is associated with decreased renal bone morphogenetic protein-7 expression in rats.
AUTHOR: Tuglular S; Gogas Yavuz D; Cakalagaoglu F; Citak L; Arikan H; Kocak H; Ozener C; Akoglu E
CORPORATE SOURCE: Section of Nephrology, Marmara University Medical School, Istanbul, Turkey.. serhantuglular@yahoo.com
SOURCE: Transplantation proceedings, (2004 Jan-Feb) Vol. 36, No. 1, pp. 131-3.
Journal code: 0243532. ISSN: 0041-1345. L-ISSN: 0041-1345.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200410
ENTRY DATE: Entered STN: 12 Mar 2004
Last Updated on STN: 6 Oct 2004
Entered Medline: 5 Oct 2004

AB The aim of our study was to investigate bone morphogenetic protein-7 (BMP-7) expression in a rat model of chronic cyclosporine (CsA) toxicity compare with healthy controls, as well as the influence of treatment with the angiotensin-converting enzyme inhibitor (ACEI) quinapril. Twenty-four male Wistar rats were divided into groups of eight animals treated with CsA (15 mg/kg intraperitoneally) for 8 weeks (CsA group) without or with quinapril (10 mg/kg per day in the drinking water: CsA group + Q) for comparison with healthy controls (H group). The renal tissues were examined by light microscopy for CsA toxicity; specifically,

tubulointerstitial damage and afferent arteriolopathy as well as BMP-7 expression were semiquantitatively scored by immunohistochemical staining. Mean CsA levels were 1982 ng/mL and 1968 ng/mL for the CsA and CsA + Q groups, respectively. At the end of the study period, the mean serum creatinine levels were 0.8 +/- 0.2 mg/dL, 1.6 +/- 0.8 mg/dL, and 1.4 +/- 0.8 mg/dL for the H, CsA, and CsA + Q groups, respectively. Interstitial fibrosis, tubular atrophy, and afferent arteriolar hyalinization were present in the CsA group and, to a lesser degree, in the CsA + Q group, compared with the H group. CsA-treated rats displayed significantly decreased BMP-7 expression compared with healthy controls (P <.0005). BMP-7 expression was higher among the CsA + Q group than the the group CsA group. In a rat model histologic changes characteristic of CsA-induced nephrotoxicity are associated with decreased expression of BMP-7, which seems to be at least partially restored by ACE inhibition.

L6 ANSWER 18 OF 48 MEDLINE on STN
ACCESSION NUMBER: 2004164940 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15058652
TITLE: In vitro acetylcholinesterase inhibition by novel OP compounds in various tissues of the fish Channa punctatus.
AUTHOR: Rahman M F; Mahboob M; Grover P
CORPORATE SOURCE: Biochemical Toxicology, Biology Division, Indian Institute of Chemical Technology, Hyderabad.
SOURCE: Bulletin of environmental contamination and toxicology, (2004 Jan) Vol. 72, No. 1, pp. 38-44.
Journal code: 0046021. ISSN: 0007-4861. L-ISSN: 0007-4861.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200405
ENTRY DATE: Entered STN: 3 Apr 2004
Last Updated on STN: 12 May 2004
Entered Medline: 11 May 2004

L6 ANSWER 19 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2003:319495 CAPLUS
DOCUMENT NUMBER: 138:343864
TITLE: In vivo delivery methods and compositions
INVENTOR(S): Kensey, Kenneth
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 45 pp., Cont.-in-part of U.S. Ser. No. 819,924.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 8
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20030078517	A1	20030424	US 2001-839785	20010420
US 6019735	A	20000201	US 1997-919906	19970828
CA 2301161	A1	19990304	CA 1998-2301161	19980826
WO 9910724	A2	19990304	WO 1998-US17657	19980826

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HU	2001000201	A2	20010528	HU 2001-201	19980826
HU	2001000201	A3	20040329		
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US	6322525	B1	20011127	US 2000-501856	20000210
NO	2000000944	A	20000225	NO 2000-944	20000225
MX	2000002073	A	20010821	MX 2000-2073	20000228
US	6428488	B1	20020806	US 2000-615340	20000712
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WO	2002043806	A2	20020606	WO 2001-US44352	20011127
WO	2002043806	A3	20030327		

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AU	2002026986	A	20020611	AU 2002-26986	20011127
US	20020088953	A1	20020711	US 2001-33841	20011227
US	6624435	B2	20030923		
WO	2002079778	A2	20021010	WO 2002-US3984	20020207
WO	2002079778	A3	20030710		

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US	20020184941	A1	20021212	US 2002-156165	20020528
US	6571608	B2	20030603		

PRIORITY APPLN. INFO.:

US 1997-919906	A2	19970828
US 1999-439795	A2	19991112
US 2000-501856	A2	20000210
US 2000-628401	A2	20000801
US 2000-727950	B2	20001201
US 2001-819924	A2	20010328
US 1997-966076	A	19971107
WO 1998-US17657	W	19980826
US 2000-615340	A3	20000712
US 2000-228612P	P	20000828
US 2001-789350	B2	20010221

US 2001-828761	A	20010409
US 2001-839785	A	20010420
US 2001-841389	A	20010424
US 2001-897164	A3	20010702
WO 2001-US44352	W	20011127

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Various methods are provided for determining and utilizing the viscosity of the circulating blood of a living being over a range of shear rates for diagnostics and treatment, such as detecting/reducing blood viscosity, work of the heart, contractility of the heart, for detecting/reducing the surface tension of the blood, for detecting plasma viscosity, for explaining/countering endothelial cell dysfunction, for providing high and low blood vessel wall shear stress data, red blood cell deformability data, lubricity of blood, and for treating different ailments such as peripheral arterial disease in combination with administering to a living being at least 1 drug. Agents effective to regulate at least 1 of the aforementioned blood parameters are used to adjust distribution of a substance through the bloodstream.

OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)

L6 ANSWER 20 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2003140252 EMBASE

TITLE: [Outcome research in cardiology. Influence of therapeutic guidelines on pharmacotherapy, morbidity and mortality in patients with hartfailure].
De invloed van behandelrichtlijnen op farmacotherapie, morbiditeit en mortaliteit bij patienten met hartfalen: Uitkomstenonderzoek in de cardiologie.

AUTHOR: De Jong, I.E.J., Dr. (correspondence); Kloeg, P.H.A.M.; Steenhoek, A.

CORPORATE SOURCE: Ziekenhuisapotheek Med. Ctr. Alkmaar, Alkmaar, Netherlands.

AUTHOR: Klungel, O.H.

CORPORATE SOURCE: Disciplinegroep F.-E./Farmacother., Universiteit Utrecht, Utrecht, Netherlands.

AUTHOR: Cornel, J.H.

CORPORATE SOURCE: Afdeling Cardiologie, Medisch Centrum Alkmaar, Alkmaar, Netherlands.

AUTHOR: De Jong, I.E.J., Dr. (correspondence)

CORPORATE SOURCE: Ziekenhuisapotheek Bovenlj Ziekenh., Postbus 37610, 1030 BD Amsterdam, Netherlands.

SOURCE: Pharmaceutisch Weekblad, (21 Mar 2003) Vol. 138, No. 12, pp. 424-429.
Refs: 8
ISSN: 0031-6911 CODEN: PHWEAW

COUNTRY: Netherlands

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 017 Public Health, Social Medicine and Epidemiology
018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index

LANGUAGE: Dutch; Flemish

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 17 Apr 2003
Last Updated on STN: 17 Apr 2003

AB Throughout the last decade, the therapeutic approach to congestive heart failure (CHF) has undergone considerable change. Several trials have indicated a beneficial effect of ACE inhibitors, beta-blocking agents, angiotensin II antagonists and spironolactone on morbidity and/or mortality in patients with CHF. The question arises whether these changes in insight of the therapeutic approach have had influence on the daily treatment of patients with CHF. The objective of

this study is to assess current trends in clinical practice in pharmacotherapy and its effect on mortality and hospitalisations for CHF, while controlling for confounding factors, at a general hospital over a 9-year period in relation to published reports and guidelines.

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YOU HAVE REQUESTED DATA FROM FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' - CONTINUE? (Y)/N:y

L6 ANSWER 21 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2003508272 EMBASE
TITLE: Emerging drugs for renal failure.
AUTHOR: Chatterjee, Prabal K., Dr. (correspondence)
CORPORATE SOURCE: Department of Pharmacology, Sch. of Pharm. and Biomol. Sciences, University of Brighton, Moulsecoomb, Brighton, BN2 4GJ, United Kingdom. p.k.chatterjee@brighton.ac.uk
AUTHOR: Thiemermann, Christoph
CORPORATE SOURCE: Dept. Exp. Med., Nephrol./Crit. Care, William Harvey Research Institute, Queen Mary - University of London, Charterhouse Square, London, EC1M 6BQ, United Kingdom. c.thiemermann@qmul.ac.uk
SOURCE: Expert Opinion on Emerging Drugs, (Nov 2003) Vol. 8, No. 2, pp. 389-435.
Refs: 374
ISSN: 1472-8214 CODEN: EOEDA3
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 22 Jan 2004
Last Updated on STN: 22 Jan 2004

AB Renal failure involves a significant impairment of the essential functions of the kidney, which can be either acute with sudden and rapid onset (acute renal failure [ARF]) or chronic with gradual onset (chronic renal failure [CRF]). ARF, if detected early, may be halted or reversed, whereas CRF is generally irreversible. Without treatment or intervention, both forms of renal failure lead to end stage renal failure (ESRF) or end stage renal disease (ESRD), requiring renal replacement therapy (RRT) in the form of dialysis or renal transplantation for survival. However, provision of RRT requires expert teams working in specialised units, making therapy of patients with renal failure expensive; furthermore, RRT is complex, with its own complications. Although pharmacological interventions have shown promise in experimental models, these have not been as successful in the clinical setting (e.g., administration of atrial natriuretic peptide, low-dose dopamine). At present, drugs are administered during CRF to either reduce one of the many risk factors of CRF (e.g., angiotensin-converting enzyme inhibitors, statins) or to deal with the consequences of CRF (e.g., erythropoietin, calcitriol). Recent evidence suggests that some of these interventions may provide further direct beneficial effects via reduction of renal inflammation. Although these interventions have greatly improved the prospects for patients suffering ESRF, the development of novel drugs and therapies with which to reduce the consequences of renal failure and ESRD remain topics of great interest.

This article reviews the therapies available for the prevention and management of renal failure in adults and describes, in detail, emerging drugs and novel interventions that may soon become available for the treatment or prevention of ESRF.

L6 ANSWER 22 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:199517 BIOSIS
DOCUMENT NUMBER: PREV200300199517
TITLE: Rationale and study design for a multicenter, randomized, double-blind, placebo-controlled study of the effects of tolvaptan on the acute and chronic outcomes of patients hospitalized with worsening congestive heart failure.
AUTHOR(S): Gheorghiade, Mihai [Reprint Author]; Gattis, Wendy A.; Barbagelata, Alejandro; Adams, Kirkwood F. Jr.; Elkayam, Uri; Orlandi, Cesare; O'Connor, Christopher M.
CORPORATE SOURCE: Division of Cardiology, Feinberg School of Medicine, Northwestern University, 201 E Huron St, Galter 10-240, Chicago, IL, 60611, USA
m-gheorghiade@northwestern.edu
SOURCE: American Heart Journal, (February 2003) Vol. 145, No. 2 Supplement, pp. S51-S54. print.
ISSN: 0002-8703 (ISSN print).
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Apr 2003
Last Updated on STN: 23 Apr 2003

L6 ANSWER 23 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2002:392219 CAPLUS
DOCUMENT NUMBER: 136:406945
TITLE: Methods for in vivo drug delivery based on monitoring blood flow parameters
INVENTOR(S): Kensey, Kenneth R.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 40 pp., Cont.-in-part of U.S. Ser. No. 727,950.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 8
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20020061835	A1	20020523	US 2001-828761	20010409
US 6019735	A	20000201	US 1997-919906	19970828
CA 2301161	A1	19990304	CA 1998-2301161	19980826
WO 9910724	A2	19990304	WO 1998-US17657	19980826
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HU 2001000201	A3	20040329		
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     NO 2000000944         A     20000225    NO 2000-944             20000225
     MX 2000002073         A     20010821    MX 2000-2073            20000228
     US 6428488            B1    20020806    US 2000-615340          20000712
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     US 6624435            B2    20030923
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     US 20020184941        A1    20021212    US 2002-156165          20020528
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                                             US 1999-439795      A2 19991112
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                                             US 2001-819924      A  20010328
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                                             US 2001-839785      A  20010420
                                             US 2001-841389      A  20010424
                                             US 2001-897164      A3 20010702
                                             WO 2001-US44352     W  20011127
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB   Various methods are provided for determining and utilizing the viscosity of the
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circulating blood of a living being over a range of shear rates for diagnostics and treatment, such as detecting/reducing blood viscosity, work of the heart, contractility of the heart, for detecting/reducing the surface tension of the blood, for detecting plasma viscosity, for explaining/countering endothelial cell dysfunction, for providing high and low blood vessel wall shear stress data, red blood cell deformability data, lubricity of blood, and for treating different ailments such as peripheral arterial disease in combination with administering to a living being at least one pharmaceutically acceptable agent. Agents pharmaceutically effective to regulate at least one of the aforementioned blood parameters are used to adjust distribution of a substance through the bloodstream.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

L6 ANSWER 24 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 2002:185688 CAPLUS
DOCUMENT NUMBER: 136:252567
TITLE: Methods for drug administration and distribution based on monitoring blood viscosity and other parameters for diagnostics and treatment
INVENTOR(S): Kensey, Kenneth
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 46 pp., Cont.-in-part of U.S. Ser. No. 819,924.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 8
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20020032149	A1	20020314	US 2001-841389	20010424
US 6019735	A	20000201	US 1997-919906	19970828
CA 2301161	A1	19990304	CA 1998-2301161	19980826
WO 9910724	A2	19990304	WO 1998-US17657	19980826
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HU 2001000201	A3	20040329		
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US 6322525	B1	20011127	US 2000-501856	20000210
NO 2000000944	A	20000225	NO 2000-944	20000225
MX 2000002073	A	20010821	MX 2000-2073	20000228
US 6428488	B1	20020806	US 2000-615340	20000712
WO 2002009583	A2	20020207	WO 2001-US23696	20010730
WO 2002009583	A3	20020425		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US,				

UZ, VN, YU, ZA, ZW, SZ, BE, CY, FR, GR, IE, IT, MC, NL, BF, BJ,
CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 20020088953	A1	20020711	US 2001-33841	20011227
US 6624435	B2	20030923		
WO 2002079778	A2	20021010	WO 2002-US3984	20020207
WO 2002079778	A3	20030710		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL,
PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG,
UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG,
KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR,
IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN,
GQ, GW, ML, MR, NE, SN, TD, TG

US 20020184941	A1	20021212	US 2002-156165	20020528
US 6571608	B2	20030603		

PRIORITY APPLN. INFO.:	US 1997-919906	A2 19970828
	US 1999-439795	A2 19991112
	US 2000-501856	A2 20000210
	US 2000-628401	A2 20000801
	US 2000-727950	A2 20001201
	US 2001-819924	A2 20010328
	US 1997-966076	A 19971107
	WO 1998-US17657	W 19980826
	US 2000-615340	A3 20000712
	US 2000-228612P	P 20000828
	US 2001-789350	B2 20010221
	US 2001-828761	A 20010409
	US 2001-839785	A 20010420
	US 2001-841389	A 20010424
	US 2001-897164	A3 20010702

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Various methods are provided for determining and utilizing the viscosity of the circulating blood of a living being, i.e., a human, over a range of shear rates for diagnostics and treatment, such as detecting/reducing blood viscosity, work of the heart, contractility of the heart, for detecting/reducing the surface tension of the blood, for detecting plasma viscosity, for explaining/countering endothelial cell dysfunction, for providing high and low blood vessel wall shear stress data, red blood cell deformability data, lubricity of blood, and for treating different ailments such as peripheral arterial disease in combination with administering to a living being at least one pharmaceutically acceptable agent. Agents pharmaceutically effective to regulate at least one of the aforementioned blood parameters are used to adjust distribution of a substance through the bloodstream. For example, when blood viscosity is a blood flow parameter monitored, an agent is selected from i.v. diluents, red blood cell deformability agents, antiurea agents, oral contraceptives, antidiabetic agents, antiarrhythmics, antihypertensives, antihyperlipidemics, antiplatelet agents, appetite suppressants, antiobesity agents, blood modifiers, smoking deterrent agents, and nutritional supplements.

OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)

L6 ANSWER 25 OF 48 MEDLINE on STN DUPLICATE 7

ACCESSION NUMBER: 2002170746 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11901939
TITLE: [Reduced risk of stroke recurrence due to hypotensive medication, irrespective of the initial blood pressure]. Minder kans op een recidiefberoerte door

bloeddrukverlagende medicatie, ongeacht de uitgangsbloeddruk.
AUTHOR: Lenders J W M; Thien Th
CORPORATE SOURCE: Universitair Medisch Centrum, afd. Algemeen Interne Geneeskunde, Postbus 9101, 6500 HB Nijmegen.. j.lenders@aig.azn.nl
SOURCE: Nederlands tijdschrift voor geneeskunde, (2002 Mar 2) Vol. 146, No. 9, pp. 398-9.
Journal code: 0400770. ISSN: 0028-2162. L-ISSN: 0028-2162.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: (CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
(ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Dutch
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200207
ENTRY DATE: Entered STN: 21 Mar 2002
Last Updated on STN: 12 Sep 2002
Entered Medline: 12 Jul 2002

AB The 'Perindopril protection against recurrent stroke study' (PROGRESS) demonstrated that for patients with a history of stroke or transient ischaemic attack during the previous 5 years, a blood-pressure-lowering regimen based on the combination of a diuretic and an angiotensin-converting enzyme (ACE) inhibitor, reduces the risk of stroke recurrence (fatal and non-fatal) by 28% (95%-CI: 17-38). This effect was irrespective of the initial neurological diagnosis (ischaemic or haemorrhagic stroke) and blood pressure level. Patients who were treated with just the ACE inhibitor did not exhibit these effects. This large-scale clinical trial demonstrates that hypotensive medication in the form of a diuretic combined with an ACE inhibitor is a beneficial strategy for the secondary prevention of stroke in normotensive and hypertensive patients.

L6 ANSWER 26 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 2003:161291 CAPLUS
DOCUMENT NUMBER: 139:255033
TITLE: New approaches to delay the progression of chronic renal failure
AUTHOR(S): Klahr, Saulo; Morrissey, Jeremiah; Hruska, Keith; Wang, Song; Chen, Qing
CORPORATE SOURCE: Departments of Internal Medicine, Cell Biology and Physiology, and Pediatrics, Washington University School of Medicine, St. Louis, MO, USA
SOURCE: Kidney International, Supplement (2002), 80, S23-S26
CODEN: KISUDF; ISSN: 0098-6577
PUBLISHER: Blackwell Science, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The mol. cloning of many bone morphogenetic proteins (BMP)-encoding genes and their identification as transforming growth factor-β (TGF-β) relatives enhanced the interest in these mols. and allowed expression and functional studies to be performed. Rats with ureteral obstruction were distributed into four groups. Group 1 received vehicle, group 2 received enalapril 12.5 mg/kg body wt/day, group 3 received 50 or 300 μg/kg body wt BMP-7, and group 4 received both enalapril and the high dose of BMP-7. We also studied the effects of BMP-7 administration in a model streptozocin-induced diabetes. Treatment with BMP-7 in rats with ureteral obstruction of 3 days duration and subsequent release indicated that this compound decreases interstitial volume and accelerates the return of renal

function. After 16 wk of diabetes, the rats were treated with BMP -7. The administration of BMP-7 partially reversed renal hypertrophy, restored GFR to normal, and decreased proteinuria. These studies indicate that administration of BMP-7 maintains and restores renal function and structure in animals with ureteral obstruction and diabetic nephropathy.

REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 27 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2001404064 EMBASE
TITLE: Potential methods to prevent interstitial fibrosis in renal disease.
AUTHOR: Strutz, F. (correspondence)
CORPORATE SOURCE: Dept. of Nephrology/Rheumatology, Georg-August-University, Robert-Koch-Str. 40, 37075 Gottingen, Germany. fstrutz@gwdg.de
SOURCE: Expert Opinion on Investigational Drugs, (2001) Vol. 10, No. 11, pp. 1989-2001.
Refs: 152
ISSN: 1354-3784 CODEN: EOIDER
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 6 Dec 2001
Last Updated on STN: 6 Dec 2001

AB Almost all forms of end stage renal disease (ESRD) are characterised by progressive interstitial fibrosis and tubular atrophy. Since most forms of chronic renal failure are initiated by inflammatory processes, anti-inflammatory strategies can be successful, if initiated early, in preventing progression of the disease process. Unfortunately, in most cases the disease is only detected clinically following robust progression of interstitial fibrosis. In these patients, control of secondary risk factors, such as hypertension and hyperglycaemia, can slow the progression rate but cannot stop the process completely. Certainly, ACE inhibitors remain the mainstay of preserving renal function. However, additional therapies are needed for the effective treatment of progressive renal fibrosis. A number of compounds have shown some very potent antifibrotic properties in vitro and in vivo, and are currently undergoing further evaluation. This review discusses the most promising among them. However, few of the therapeutic agents discussed here have been tested clinically. Studies evaluating the potential of a number of these have just commenced whereas for many others clinical use is still many years away. However, some very promising reagents may enhance our clinical arsenal within a relatively short period of time.

L6 ANSWER 28 OF 48 MEDLINE on STN DUPLICATE 8

ACCESSION NUMBER: 2001339301 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11245776
TITLE: Urinary tract obstruction.
AUTHOR: Klahr S
CORPORATE SOURCE: Department of Medicine, Barnes-Jewish Hospital at Washington University School of Medicine, St. Louis, MO 63110-1092, USA.. sklahr@imgate.wustl.edu
SOURCE: Seminars in nephrology, (2001 Mar) Vol. 21, No. 2, pp. 133-45. Ref: 84
Journal code: 8110298. ISSN: 0270-9295. L-ISSN: 0270-9295.

PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 18 Jun 2001
Last Updated on STN: 18 Jun 2001
Entered Medline: 14 Jun 2001

AB Angiotensin II plays a pivotal role in the progression of renal diseases, including obstructive nephropathy. Increasing levels of angiotensin II in obstructive nephropathy upregulate the expression of several factors: transforming growth factor-beta1 (TGF-beta1), tumor necrosis factor-alpha (TNF-alpha), platelet-derived growth factor (PDGF), insulin-like growth factor (IGF-1), osteopontin, vascular cell adhesion molecule-1 (VCAM-1), nuclear factor-kappaB (NF-kappaB), monocyte chemoattractant peptide-1 (MCP-1), intercellular adhesion molecule-1 (ICAM-1), and CD14 among others. Local production of TGF-beta, by intrinsic renal cells or by macrophages invading the kidney, is a key mediator of renal fibrosis. Activation of TGF-beta stimulates endothelin production. Endothelin, in turn, is a potent stimulus for fibrogenesis. Oxidative stress, fueled in part by angiotensin II, upregulates the expression of adhesion molecules, chemoattractant compounds and cytokines. Sustained obstructive nephropathy leads to apoptosis of tubular epithelial cells. Several factors and genes involved in apoptosis have been described. Nuclear factor kappa-B is involved in the transcriptional regulation of genes present in several organs, including the kidney. NF-kappaB is activated in the setting of ureteral obstruction. Administration of angiotensin-converting enzyme (ACE) inhibitors decreased significantly the activation of NF-kappaB in the obstructed kidney. Studies in neonatal rats indicate that chronic ureteral obstruction decreases the expression of epidermal growth factor (EGF). Replacement of this factor decreased apoptosis and reduced the expression of vimentin, clusterin, and TGF-beta. The administration of IGF-1 also lessened the tubular and interstitial pathology in the setting of ureteral obstruction. A spectrum of urinary tract malformations have been described. The utility of certain markers such as fetal serum beta(2) microglobulin as a predictor of postnatal renal function in bilateral uropathies has been described. A number of pharmacologic interventions that ameliorate the increased expansion of the interstitial volume, decrease the expression of TGF-beta, and down-regulate the production of extracellular matrix and the infiltration of the interstitium by macrophages have been described. Drugs used include ACE inhibitors, administration of arginine, administration of osteogenic protein-1, Pirferidone, and so on. It is likely that in the next decade advances in genetic manipulations and new drug therapies may forestall the development of fibrosis in the setting of urinary tract obstruction.

L6 ANSWER 29 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2000417911 EMBASE
TITLE: [The protective role of ace inhibitors on the kidney function].
De beschermende rol van ACE-remmers op de nierfunctie.
AUTHOR: Lameire, N., Dr. (correspondence)
CORPORATE SOURCE: Nefrologie, Universitair Ziekenhuis, Ziekenhuis Gent, Gent, Belgium.
SOURCE: Tijdschrift voor Geneeskunde, (15 Nov 2000) Vol. 56, No. 22, pp. 1661-1662.

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                     Refs: 7
                     ISSN: 0371-683X  CODEN: TGEKBW
COUNTRY:             Belgium
DOCUMENT TYPE:       Journal; Note
FILE SEGMENT:        028     Urology and Nephrology
                     003     Endocrinology
                     030     Clinical and Experimental Pharmacology
                     037     Drug Literature Index
                     006     Internal Medicine
LANGUAGE:            Dutch; Flemish
ENTRY DATE:          Entered STN: 14 Dec 2000
                     Last Updated on STN: 14 Dec 2000

L6   ANSWER 30 OF 48  EMBASE  COPYRIGHT (c) 2010 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    2000229712  EMBASE
TITLE:               [Advantage ACE inhibitors. The effect
                     of RAAS inhibitors on endothelial function].
                     De invloed van RAAS-remmers op de
                     endotheelfunctie. Een pluspunt voor de ACE-remmers.
AUTHOR:              Huisman, A. (correspondence); Voorbij, H.A.M.
CORPORATE SOURCE:    Centraal Diagnostisch Laboratorium G.03.550, Universitair
                     Medisch Centrum Utrecht, Postbus 85.500, 3508 GA Utrecht.
AUTHOR:              Huisman, A. (correspondence)
CORPORATE SOURCE:    Centraal Diagnostisch Lab. G.03.550, Universitair Medisch
                     Centrum Utrecht, Postbus 85.500, 3508 GA Utrecht,
                     Netherlands.
SOURCE:              Pharmaceutisch Weekblad, (23 Jun 2000) Vol. 135, No. 25,
                     pp. 906-910.
                     Refs: 25
                     ISSN: 0031-6911  CODEN: PHWEAW
COUNTRY:             Netherlands
DOCUMENT TYPE:       Journal; Article
FILE SEGMENT:        018     Cardiovascular Diseases and Cardiovascular Surgery
                     030     Clinical and Experimental Pharmacology
                     037     Drug Literature Index
LANGUAGE:            Dutch; Flemish
SUMMARY LANGUAGE:    English; Dutch; Flemish
ENTRY DATE:          Entered STN: 20 Jul 2000
                     Last Updated on STN: 20 Jul 2000
AB   Atherosclerosis is accompanied by endothelial dysfunction.  Nitric oxide,
     produced by the endothelial cell, has an antiatherogenic and
     antithrombotic effect.  ACE inhibitors have, by
     limiting the degradation of bradykinin, a beneficial effect on the
     production of nitric oxide.  It has been suggested from the outcomes of
     the TREND and SAVE studies that ACE inhibitors may
     have an antithrombotic and antiatherogenic effect.  Angiotensin II
     antagonists possibly lack this beneficial effect of ACE
     inhibitors.


=> dis ibib abs l6 31-40
YOU HAVE REQUESTED DATA FROM FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' - CONTINUE?
(Y)/N:Y



L6   ANSWER 31 OF 48     MEDLINE on STN
ACCESSION NUMBER:    2000403356     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 10894795
TITLE:               Osteogenic protein-1 prevents renal
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fibrogenesis associated with ureteral obstruction.
AUTHOR: Hruska K A; Guo G; Wozniak M; Martin D; Miller S; Liapis H; Loveday K; Klahr S; Sampath T K; Morrissey J
CORPORATE SOURCE: Renal Division, Departments of Medicine, Barnes-Jewish Hospital at Washington University, St. Louis, Missouri 63110, USA.. khruska@imgate.wustl.edu
CONTRACT NUMBER: AR-32087 (United States NIAMS NIH HHS)
AR-39561 (United States NIAMS NIH HHS)
P01-DK-09976 (United States NIDDK NIH HHS)
SOURCE: American journal of physiology. Renal physiology, (2000 Jul) Vol. 279, No. 1, pp. F130-43.
Journal code: 100901990. ISSN: 0363-6127. L-ISSN: 0363-6127.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 1 Sep 2000
Last Updated on STN: 10 Dec 2002
Entered Medline: 23 Aug 2000

AB Unilateral ureteral obstruction (UUO) is a model of renal injury characterized by progressive tubulointerstitial fibrosis and renal damage, while relatively sparing the glomerulus and not producing hypertension or abnormalities in lipid metabolism. Tubulointerstitial fibrosis is a major component of several kidney diseases associated with the progression to end-stage renal failure. Here we report that when a critical renal developmental morphogen, osteogenic protein-1 (OP-1; 100 or 300 microg/kg body wt), is administered at the time of UUO and every other day thereafter, interstitial inflammation and fibrogenesis are prevented, leading to preservation of renal function during the first 5 days after obstruction. Compared with angiotensin-converting enzyme inhibition with enalapril treatment, OP-1 was more effective in preventing tubulointerstitial fibrosis and in preserving renal function. The mechanism of OP-1-induced renal protection was associated with prevention of tubular atrophy, an effect not shared with enalapril, and was related to preservation of tubular epithelial integrity. OP-1 blocked the stimulation of epithelial cell apoptosis produced by UUO, which promoted maintenance of tubular epithelial integrity. OP-1 preserved renal blood flow (RBF) during UUO, but enalapril also stimulated RBF. Thus OP-1 treatment inhibited tubular epithelial disruption stimulated by the renal injury of UUO, preventing tubular atrophy and diminishing the activation of tubulointerstitial inflammation and fibrosis and preserving renal function.

L6 ANSWER 32 OF 48 EMBASE
ACCESSION NUMBER: 1999201622 EMBASE
TITLE: [The effect on mortality is still not clear. The prominent place of diuretics in congestive heart failure].
De prominente rol van diuretica. Het effect op de overleving blijft onduidelijk.
AUTHOR: Dormans, T.P.J., Dr. (correspondence)
AUTHOR: Dormans, T.P.J., Dr. (correspondence)
CORPORATE SOURCE: Afdeling Intensive Care, Atrium Medisch Centrum, Postbus 4446, 6401 CX Heerlen, Netherlands.
SOURCE: Pharmaceutisch Weekblad, (4 Jun 1999) Vol. 134, No. 22, pp. 758-761.
Refs: 23

ISSN: 0031-6911 CODEN: PHWEAW
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
LANGUAGE: Dutch; Flemish
SUMMARY LANGUAGE: English; Dutch; Flemish
ENTRY DATE: Entered STN: 1 Jul 1999
Last Updated on STN: 1 Jul 1999

AB Forty years after their introduction diuretics still form one of the cornerstones in the treatment of chronic heart failure. Due to a prompt positive effect on the symptoms, their role on the quality of life is beyond doubt. On the other side, their effects on the progression of heart failure are less clear. To avoid any negative effects on the latter, diuretics should be used in combination with ACE-inhibitors whenever possible. The emphasis in this article lies on the diuretic treatment of patients who are unresponsive to the usual diuretic regimen.

L6 ANSWER 33 OF 48 MEDLINE on STN DUPLICATE 9
ACCESSION NUMBER: 1999254560 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10321257
TITLE: [Eisenmenger syndrome in adults].
Het Eisenmenger-syndroom op volwassen leeftijd.
AUTHOR: Roos-Hesselink J W; Meijboom F J; Spitaels S E
CORPORATE SOURCE: Thoraxcentrum, Academisch Ziekenhuis Rotterdam-Dijkzigt.
SOURCE: Nederlands tijdschrift voor geneeskunde, (1999 Mar 6) Vol. 143, No. 10, pp. 501-5. Ref: 22
Journal code: 0400770. ISSN: 0028-2162. L-ISSN: 0028-2162.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: Dutch
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 14 Jun 1999
Last Updated on STN: 14 Jun 1999
Entered Medline: 1 Jun 1999

AB In Eisenmenger's syndrome a central left-to-right shunt in the heart, a congenital anomaly, leads to pulmonary hypertension which subsequently causes the shunt to be reversed. The hypoxaemia resulting from a right-to-left shunt is compensated by an increase of the haemoglobin concentration due to a rise of the haematocrit. In adult patients not operated (adequately), the symptoms are the consequence of the erythrocytaemia and an increased haemorrhagic diathesis. In the long run heart failure develops. Phlebotomy is indicated for patients with haematocrits higher than 0.65 with signs of hyperviscosity and is also advised before non-cardiac surgery to improve coagulation parameters. Phlebotomy should be performed slowly (500 ml in 30-45 min) with simultaneous volume replacement. Excessive phlebotomy causes iron deficiency and spherocytosis which increase viscosity as well as the risk of CVA. Treatment consists of iron supplementation. Anticoagulation is indicated only in case of atrial fibrillation or mechanical valves. The use of acetylsalicylacid or NSAIDs is relatively contraindicated, because of abnormal haemostasis in these patients. During treatment with ACE inhibitors and other vasodilators, hypovolaemia should be avoided, because at a lower systemic blood pressure the right-to-left shunt increases and a potentially fatal cyanosis may occur.

L6 ANSWER 34 OF 48 EMBASE

ACCESSION NUMBER: 1999120434 EMBASE
TITLE: [What is increased, what is decreased. Transmural investigation of prescription behaviour: The effects of hospitalisation on drug utilization].
Transmuraal prescriptieonderzoek: De effecten van een ziekenhuisopname op de medicatie. Wat kwam erbij, wat ging eraf.
AUTHOR: Feberwee, T. (correspondence); Meulepas, M.; Brenninkmeijer, V.J.; Verstappen, W.H.J.M.
AUTHOR: Feberwee, T. (correspondence)
CORPORATE SOURCE: Apotheek Reinier de Graaf Groep, Postbus 5011, 2600 GA Delft, Netherlands.
SOURCE: Pharmaceutisch Weekblad, (19 Mar 1999) Vol. 134, No. 11, pp. 393-397.
Refs: 13
ISSN: 0031-6911 CODEN: PHWEAW
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 017 Public Health, Social Medicine and Epidemiology
037 Drug Literature Index
006 Internal Medicine
LANGUAGE: Dutch; Flemish
SUMMARY LANGUAGE: English; Dutch; Flemish
ENTRY DATE: Entered STN: 22 Apr 1999
Last Updated on STN: 22 Apr 1999

AB The effects of hospitalisation on the medication were analysed in Sint Joseph's Hospital in Veldhoven. Patients admitted to an internal medicine ward, a cardiology ward and a pulmonology ward, ovre a period of two months, were included. Medication schemes at admission, at discharge and six months after discharge of 184 patients were retrospectively compared. Mean numbers of prescriptions per patient did not change between admission and discharge (5.8 versus 5.9). 36% of the admission medication was discontinued and 36% of the discharge medication was started in hospital. Main changes in prescriptions occurred in the following medication groups: an increase of H2- inhibitors and proton pump inhibitors (40%), of xanthine derivatives (24%) and of ACE-inhibitors (23%) and a decrease in prescriptions of corticosteroids for inhalation (38%) and of NSAID's (24%).

L6 ANSWER 35 OF 48 MEDLINE on STN
ACCESSION NUMBER: 1999164651 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10065257
TITLE: [Immunosuppressive treatment of patients with a nephrotic syndrome due to minimal change glomerulopathy].
Immunosuppressieve behandeling van patienten met een nefrotisch syndroom op basis van 'minimal-change'-glomerulopathie.
AUTHOR: Branten A J; Wetzels J F
CORPORATE SOURCE: Academisch Ziekenhuis, afd. Nierziekten, Nijmegen.
SOURCE: Nederlands tijdschrift voor geneeskunde, (1998 Dec 26) Vol. 142, No. 52, pp. 2832-8. Ref: 31
Journal code: 0400770. ISSN: 0028-2162. L-ISSN: 0028-2162.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: Dutch
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 26 Apr 1999
Last Updated on STN: 26 Apr 1999
Entered Medline: 13 Apr 1999

AB Prednisone monotherapy is the treatment of choice for patients with a nephrotic syndrome due to minimal change glomerulopathy. In adult patients treatment should be continued for at least 24 weeks. Within this period a remission of proteinuria will occur in 75 to 90% of the patients. Patients who do not respond satisfactorily to prednisone treatment can be treated with alkylating agents. Cyclophosphamide is the drug used most commonly. Prolonged treatment (> 12 weeks) is associated with a high risk of infertility. If alkylating agents cannot be used, prolonged treatment with ciclosporine is an option.

L6 ANSWER 36 OF 48 MEDLINE on STN
ACCESSION NUMBER: 1998212129 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9550772
TITLE: [Effect of protein restriction on deterioration of kidney function].
Het effect van eiwitbeperking op nierfunctieachteruitgang.
AUTHOR: Gansevoort R T; de Zeeuw D; de Jong P E
CORPORATE SOURCE: Academisch Ziekenhuis, afd. Inwendige Geneeskunde, Groningen.
SOURCE: Nederlands tijdschrift voor geneeskunde, (1997 Nov 1) Vol. 141, No. 44, pp. 2106-10. Ref: 28
Journal code: 0400770. ISSN: 0028-2162. L-ISSN: 0028-2162.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Commentary
(ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: Dutch
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 8 Jul 1998
Last Updated on STN: 29 Jan 1999
Entered Medline: 19 Jun 1998
AB The results of meta-analyses and of the recent 'modification of diet in renal disease' study show that dietary protein restriction slows down deterioration of renal function in patients with a moderately impaired renal function. However, this effect appears to be less pronounced than previously assumed. Protein restriction probably leads to narrowing of the preglomerular blood vessel, which reduces the raised intraglomerular pressure. The resulting decrease in the filtering glomerular pressure initially leads to a slight loss of renal function, but there after to a decelerated deterioration of renal function. The aim should be a protein intake of 0.6-0.75 g per kg per day. Dietary compliance should not be predicted anamnestically but should be calculated according to the (modified) Maroni equation (protein intake = (0.18 x urinary urea excretion in mmol per 24 hours) + 15 + urinary protein excretion in g per 24 hours). This kind of protein restriction as a rule does not lead to nutritional deficiencies.

L6 ANSWER 37 OF 48 MEDLINE on STN
ACCESSION NUMBER: 1996242842 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8684502
TITLE: [ACE-inhibitors increase the risk of hypoglycemia in diabetics].
ACE-remmers vergroten het risico op hypoglykemie bij diabetici.
AUTHOR: Gansevoort R T; Sluiter W J
SOURCE: Nederlands tijdschrift voor geneeskunde, (1996 May 11) Vol. 140, No. 19, pp. 1042-4.
Journal code: 0400770. ISSN: 0028-2162. L-ISSN: 0028-2162.
PUB. COUNTRY: Netherlands

DOCUMENT TYPE: Letter
LANGUAGE: Dutch
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199608
ENTRY DATE: Entered STN: 28 Aug 1996
Last Updated on STN: 6 Feb 1998
Entered Medline: 22 Aug 1996

L6 ANSWER 38 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1996153559 EMBASE
TITLE: [ACE inhibitors increase the risk of hypoglycemia in diabetic patients [2]].
ACE-REMMERS VERGROTEN HET RISICO OP HYPOGLYKEMIE BIJ DIABETICI [2].
AUTHOR: Gansevoort, R.T.; Sluiter, W.J.; Herings, R.M.C.; De Boer, A.; Stricker, B.H.C.; Leufkens, H.G.M.; Porsius, A.
SOURCE: Nederlands Tijdschrift voor Geneeskunde, (1996) Vol. 140, No. 19, pp. 1042-1044.
ISSN: 0028-2162 CODEN: NETJAN
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Letter
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
003 Endocrinology
037 Drug Literature Index
LANGUAGE: Dutch; Flemish
ENTRY DATE: Entered STN: 3 Jul 1996
Last Updated on STN: 3 Jul 1996

L6 ANSWER 39 OF 48 MEDLINE on STN DUPLICATE 10
ACCESSION NUMBER: 1996325281 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8671960
TITLE: Effect of ramipril, nifedipine, and moxonidine on glomerular morphology and podocyte structure in experimental renal failure.
AUTHOR: Amann K; Nichols C; Tornig J; Schwarz U; Zeier M; Mall G; Ritz E
CORPORATE SOURCE: Departments of Pathology Heidelberg and Darmstadt, Department of Internal Medicine Ruperto Carola University, Heidelberg, Germany.
SOURCE: Nephrology, dialysis, transplantation : official publication of the European Dialysis and Transplant Association - European Renal Association, (1996 Jun) Vol. 11, No. 6, pp. 1003-11.
Journal code: 8706402. ISSN: 0931-0509. L-ISSN: 0931-0509.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199612
ENTRY DATE: Entered STN: 28 Jan 1997
Last Updated on STN: 28 Jan 1997
Entered Medline: 17 Dec 1996
AB BACKGROUND: Experimental renal failure causes structural alterations of the kidney. It is still unresolved how these changes are modified by antihypertensive treatment. Purpose of the study. To examine the effects of different antihypertensive agents (ramipril, nifedipine, moxonidine) mainly on glomerular geometry, cell number, cell morphology, and capillarization, in a subtotal nephrectomy model of renal failure. MATERIAL AND METHODS: Sham-operated male SD rats and subtotally nephrectomized (SNX) ad libitum-fed rats were examined. Groups of 8-10

SNX rats were left untreated or were treated with ramipril (0.5 mg/kg b.w. per day), nifedipine (20 mg/kg b.w. per day) or moxonidine (10 mg/kg b.w.per day) respectively. After perfusion fixation the kidneys were examined using stereological techniques. RESULTS: Systolic blood pressure (by tail plethysmography) was 110+/-13 mm Hg in sham-op and 119+/-9 in SNX. It was effectively and comparably reduced below normal values by ramipril (89+/-11 mmHg), nifedipine (98+/-23 mmHg) and moxonidine (92+/-11 mmHg). The glomerulosclerosis index (SI) was significantly increased in SNX versus sham-op; it was similarly decreased by ramipril and moxonidine but less so by nifedipine. Vascular damage (preglomerular vessels) was reduced by all treatments whereas tubulointerstitial damage was signficantly reduced only by ramipril and moxonidine. Mean glomerular tuft volume was increased in SNX compared to sham-op. controls and was normalized only by ramipril treatment. Glomerular cells were differentially affected the three antihypertensive agents. After subtotal nephrectomy an increase in podocyte volume and mesangial cell number per glomerulus was noted. Nifedipine, and to a lesser extent ramipril, prevented mesangial cell hyperplasia. In contrast, only the ACE inhibitor ramipril, but not nifedipine or moxonidine prevented podocyte abnormalities, particularly podocyte hypertrophy. CONCLUSIONS: (i) Despite comparable reduction in systolic blood pressure, different classes of antihypertensive agents had diverse effects on renal damage in subtotally nephrectomized rat. This observation is consistent with specific, non-hemodynamic actions of anti-hypertensives. (ii) Glomerular and tubulointerstitial damage are prevented by treatment with ACE inhibitors and antisympathotonic agents, but not with the calcium antagonist nifedipine. In contrast, renal vascular changes were also prevented by nifedipine. (iii) Only ACE inhibitors effectively inhibited podocyte hypertrophy and mesangial cell hyperplasia. Whether the superior effect of ACE inhibitors on glomerulosclerosis is related to inhibition of glomerular growth and podocyte hypertrophy as well as preservation of podocyte structure, or whether these findings are merely a passive reflection of greater efficacy, remains unresolved.

L6 ANSWER 40 OF 48 EMBASE

ACCESSION NUMBER: 1996063735 EMBASE

TITLE: [ACE inhibitors increase the risk of hypoglycaemia in diabetics].
ACE-REMMERS VERGROTEN HET RISICO OP HYPOGLYKEMIE BIJ DIABETICI.

AUTHOR: Herings, R.M.C., Dr. (correspondence); De Boer, A.; Leufkens, H.G.M.; Porsius, A.J.
CORPORATE SOURCE: Universiteit, Utrecht Inst. Pharmaceutical Sci., Sectie Farmaco-Epidemiol. F., Postbus 80.082, 3508 TB Utrecht, Netherlands.
AUTHOR: Stricker, B.H.Ch.
CORPORATE SOURCE: Acad. Ziekenhuis Rotterdam-Dijkzigt, Afd. Interne Geneeskunde II, Rotterdam, Netherlands.
AUTHOR: Herings, R.M.C., Dr. (correspondence)
CORPORATE SOURCE: Universiteit Utrecht, Utrecht Inst. Pharmaceutical Scis., Sectie Farm.-Epidemiol./Farmacother., Postbus 80.082, 3508 TB Utrecht, Netherlands.

SOURCE: Nederlands Tijdschrift voor Geneeskunde, (24 Feb 1996) Vol. 140, No. 8, pp. 432-436.
Refs: 20
ISSN: 0028-2162 CODEN: NETJAN
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
003 Endocrinology

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                    037     Drug Literature Index
                    038     Adverse Reactions Titles
LANGUAGE:           Dutch; Flemish
SUMMARY LANGUAGE:   Dutch; Flemish; English
ENTRY DATE:         Entered STN: 19 Mar 1996
                    Last Updated on STN: 19 Mar 1996
AB   Objective.  To determine the association of hypoglycaemia in diabetic
     patients with the use of ACE inhibitors.  Design.
     Nested case control study.  Setting.  Six medium sized Dutch cities
     (300.000 inhabitants).  Method.  Using data in the Dutch PHARMO system
     (1986-1992) 94 diabetic patients treated with insulin or with oral
     antidiabetic drugs, who had been admitted to hospital with hypoglycaemia,
     were identified.  From the same cohort 654 control patients were selected
     at random.  Results.  With adjustment for a wide range of potential
     confounding factors, hypoglycaemia was significantly associated with
     current use of ACE inhibitors (odds ratio: 2.8 (95% CI
     1.4-5.7)).  Both among users of insulin and users of oral antidiabetic
     drugs, use of ACE inhibitors was significantly
     associated with an increased risk of hospital admission for hypoglycaemia
     (2.8 (1.2-6.4) and 4.1 (1.4-12.2), respectively).  It was estimated that
     about 13.8% of all admissions for hypoglycaemia in diabetic patients were
     due to the use of ACE inhibitors.  Conclusion.
     Although the use of ACE inhibitors for hypertension in
     diabetic patients has advantages over the use of other antihypertensive
     drugs, one should be aware of the increased risk of hypoglycaemia.


=> dis his

     (FILE 'HOME' ENTERED AT 22:23:59 ON 05 MAY 2010)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 22:24:18 ON 05 MAY 2010
L1         88349 S (ACE(W)INHIBITOR OR ANGIOTENSIN(W)CONVERTING(W)ENZYME(W)INHIB
L2            66 S L1 AND (BONE(W)MORPHOGEN OR OSTEOGENIC(W)PROTEIN OR BMP OR OP
L3          5814 S L1 AND PROTEINURIA
L4             6 S L2 AND PROTEINURIA
L5             6 DUP REM L4 (0 DUPLICATES REMOVED)
L6            48 DUP REM L2 (18 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 22:28:10 ON 05 MAY 2010

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 22:30:34 ON 05 MAY 2010

     FILE 'STNGUIDE' ENTERED AT 22:30:35 ON 05 MAY 2010

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 22:32:19 ON 05 MAY 2010

     FILE 'STNGUIDE' ENTERED AT 22:32:21 ON 05 MAY 2010

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 22:34:07 ON 05 MAY 2010

     FILE 'STNGUIDE' ENTERED AT 22:34:09 ON 05 MAY 2010

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 22:41:37 ON 05 MAY 2010

     FILE 'STNGUIDE' ENTERED AT 22:41:40 ON 05 MAY 2010

=> dis ibib abs l6 41-48
YOU HAVE REQUESTED DATA FROM FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' - CONTINUE?
(Y)/N:y
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L6 ANSWER 41 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1996108863 EMBASE
TITLE: [ACE inhibitors after acute heart infarction: Whom, when and which?]. ACE-REMMERS NA EEN ACUUT MYOCARD-INFARCT: BIJ WIE, OP WELK MOMENT EN MET WELK MIDDEL?.
AUTHOR: Voors, A.A. (correspondence); Kingma, J.H.; Van Gilst, W.H.
CORPORATE SOURCE: Afd. Klinische Farmacologie, Rijks Universiteit, Groningen, Netherlands.
SOURCE: Hart Bulletin, (1996) Vol. 27, No. 2, pp. 51-56.
ISSN: 0301-8202 CODEN: HTBUA5
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
LANGUAGE: Dutch; Flemish
SUMMARY LANGUAGE: Dutch; Flemish
ENTRY DATE: Entered STN: 20 May 1996
Last Updated on STN: 20 May 1996

L6 ANSWER 42 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

ACCESSION NUMBER: 1994:2264 BIOSIS
DOCUMENT NUMBER: PREV199497015264
TITLE: Effects of angiotensin-converting enzyme inhibitors and prostaglandin E-1 derivatives on the cardiomyopathic hamster.
AUTHOR(S): Kato, Mitsutoshi; Yang, Jie; Iwai, Takaaki; Tanamura, Akira; Takeda, Nobuakira; Nagano, Makoto
CORPORATE SOURCE: Dep. Intern. Med., Aoto Hosp., Jikei Univ. Sch. Med., 6-41-2 Aoto, Katsushika-ku, Tokyo 125, Japan
SOURCE: Nagano, M. [Editor]; Takeda, N. [Editor]; Dhalla, N. S. [Editor]. (1994) pp. 157-164. The cardiomyopathic heart. Publisher: Raven Press, 1185 Avenue of the Americas, New York, New York 10036-2806, USA.
Meeting Info.: Symposium. Tokyo, Japan. May 1992.
ISBN: 0-7817-0092-2.
DOCUMENT TYPE: Book
Conference; (Meeting)
Book; (Book Chapter)
Conference; (Meeting Paper)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Jan 1994
Last Updated on STN: 5 Mar 1994

L6 ANSWER 43 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 1994:51514 CAPLUS
DOCUMENT NUMBER: 120:51514
ORIGINAL REFERENCE NO.: 120:9399a,9402a
TITLE: Effects of angiotensin-converting enzyme inhibitors and prostaglandin E1 derivatives on the cardiomyopathic hamster
AUTHOR(S): Kato, Mitsutoshi; Yang, Jie; Iwai, Takaaki; Tanamura, Akira; Takeda, Nobuakira; Nagano, Makoto
CORPORATE SOURCE: Sch. Med., Jikei Univ., Tokyo, 125, Japan
SOURCE: Cardiomyopathic Heart (1994), 157-64. Editor(s): Nagano, Makoto; Takeda, Nobuakira; Dhalla, Naranjan S. Raven: New York, N. Y.
CODEN: 59OTAH

DOCUMENT TYPE: Conference
LANGUAGE: English
AB Captopril or enalapril was administered to 5-wk-old J-2-N cardiomyopathic hamsters and the roles of the renin-angiotensin-aldosterone (RAA) and kallikrein-kinin systems in the onset and progress of cardiomyopathy were examined OP-1206-αCD, a prostaglandin E1 derivative was also administered. In the untreated group, serum creatine kinase levels increased in accordance with the progression of cardiomyopathy, but this increase was markedly inhibited by the administration of captropil. The rise in serum aldolase levels was similarly inhibited. Serum malondialdehyde (MDA) levels were significantly reduced by the administration of captopril. Electrocardiog. findings and the ventricular myosin isoenzyme patterns were also markedly improved by captopril. The improvement in all these parameters was less with administration of enalapril. These differences between captopril and enalapril suggest that increases in tissue bradykinin and vasodilatory prostaglandins may play an important role in the beneficial effects of captopril. When OP -1206-αCD was administered to J-2-N hamsters, some tendency of beneficial effects was observed These results suggest the importance of the kallikrein-kinin system in the development of cardiomyopathy in J-2-N cardiomyopathic hamsters.

L6 ANSWER 44 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1993222896 EMBASE
TITLE: [Quality of life during antihypertensive treatment with bisoprolol and enalapril].
BISOPROLOL VERSUS ENALAPRIL; EEN DUBBELBLIND VERGELIJKEND ONDERZOEK NAAR DE INVLOED OP THE KWALITEIT VAN HET LEVEN.
AUTHOR: Van Bortel, L.M.A.B. (correspondence); Ciampricotti, R.; Tromp, G.P.; Valster, F.A.; Lageweg, E.; Joosten, J.; Lustermans, F.A.T.; Breed, J.G.S.
CORPORATE SOURCE: Vakgroep Farmacologie, Rijksuniversiteit Limburg, Postbus 616, 6200 MD Maastricht, Netherlands.
SOURCE: TGO - Tijdschrift voor Therapie Geneesmiddel en Onderzoek, (1993) Vol. 18, No. 5, pp. 151-155.
ISSN: 0921-562X CODEN: TTTOE9
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
006 Internal Medicine
LANGUAGE: Dutch; Flemish
SUMMARY LANGUAGE: English; Dutch; Flemish
ENTRY DATE: Entered STN: 29 Aug 1993
Last Updated on STN: 29 Aug 1993
AB The well-being of hypertensive patients (expressed as 'quality of life') may be adversely affected by antihypertensive therapy. Some recently developed antihypertensive agents have been suggested to be devoid of these deleterious effects. In a double-blind cross-over study, the effect on blood pressure and quality of life of the angiotensin converting enzyme (ACE) inhibitor enalapril was compared with the effect of the highly selective β1-adrenoreceptor antagonist bisoprolol. The antihypertensive effect was at least as good with bisoprolol as with enalapril. Quality of life as measured with the Inventory of Subjective Health was comparable for the two drugs. Spontaneously mentioned adverse effects were more frequent (74%) during enalapril than during bisoprolol treatment. At the end of the study, 69% of patients chose to continue antihypertensive treatment with bisoprolol. This study shows that

bisoprolol is at least as effective as enalapril as for as antihypertensive effect and quality of life are concerned.

L6 ANSWER 45 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1993185399 EMBASE
TITLE: [New possibilities in screening for renovascular hypertension].
NIEUWE MOGELIJKHEDEN VOOR HET SCREENEN OP RENOVASCULAIRE HYPERTENSIE.
AUTHOR: Huisman, R.M., Dr. (correspondence); Jonker, G.J.
CORPORATE SOURCE: Rijksuniversiteit, Faculteit der Geneeskunde, Vakgroep Inwendige Geneeskunde, Oostersingel 59, 9713 EZ Groningen, Netherlands.
SOURCE: TGO - Tijdschrift voor Therapie Geneesmiddel en Onderzoek, (1993) Vol. 18, No. 4, pp. 106-110.
ISSN: 0921-562X CODEN: TTTOE9
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
LANGUAGE: Dutch; Flemish
SUMMARY LANGUAGE: English; Dutch; Flemish
ENTRY DATE: Entered STN: 25 Jul 1993
Last Updated on STN: 25 Jul 1993

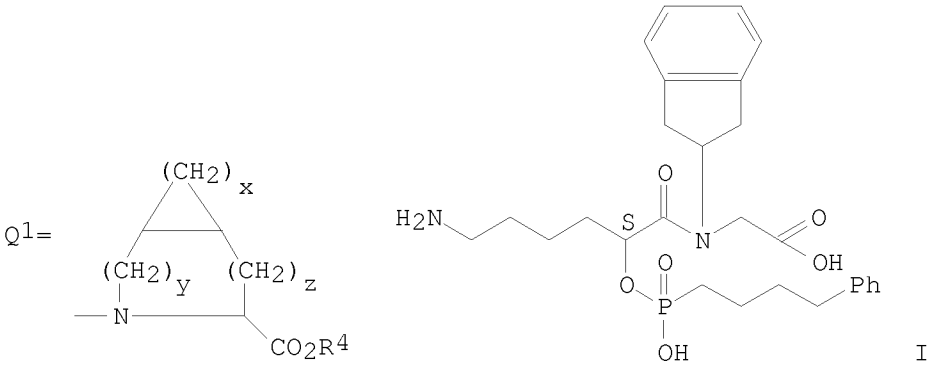
AB The use of ACE-inhibitors has resulted in improved screening methods for renovascular hypertension. The best screening test appears to be captopril renography, with a sensitivity and specificity for renovascular hypertension both of around 90% if an isotope renogram is done after a single dose of oral captopril. Another possibility, the 'captopril test', in which the plasma renin activity is determined after a single gift of captopril, is slightly less reliable, especially with comoromised renal function.

L6 ANSWER 46 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN

ACCESSION NUMBER: 1991:536776 CAPLUS
DOCUMENT NUMBER: 115:136776
ORIGINAL REFERENCE NO.: 115:23479a,23482a
TITLE: Preparation of phosphonate-substituted amino or imino acids useful as antihypertensives
INVENTOR(S): Karanewsky, Donald S.
PATENT ASSIGNEE(S): E. R. Squibb and Sons, Inc., USA
SOURCE: Eur. Pat. Appl., 46 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
EP 414190	A2	19910227	EP 1990-115930		19900820
EP 414190	A3	19910717			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE					
US 5102875	A	19920407	US 1990-542504		19900625
AU 9059105	A	19910221	AU 1990-59105		19900718
AU 632596	B2	19930107			
CA 2021409	A1	19910222	CA 1990-2021409		19900718
JP 03090091	A	19910416	JP 1990-222313		19900821
US 5436245	A	19950725	US 1993-153500		19931117
PRIORITY APPLN. INFO.:			US 1989-396170	A	19890821
			US 1992-896912	B1	19920611

OTHER SOURCE(S): MARPAT 115:136776
GI



AB R1(R3O)P(O)OCHR2COX [X = NR4CHR6CO2R4, Q1; R1 = alkyl, aminoalkyl, haloalkyl, furyl, furylalkyl, thienyl, thienylallyl, cycloalkyl, cycloalkylalkyl, pyridyl, pyridylalkyl, (substituted) Ph, phenylalkyl, acylaminomethyl; R2 = H, (halo)alkyl, (hydroxy)phenylalkyl, 2-imidazolylalkyl, aminoalkyl, 3-indolylalkyl, mercaptoalkyl, alkylthioalkyl, guanidinoalkyl, carbamoylalkyl; R3, R4 = H, alkyl, PhCH2, Ph2CH, alkali metal, acycloxymethyl; R5 = substituted phenylalkyl, methylenedioxyphenylalkyl, benzocycloalkyl; R6 = R2 except haloalkyl; x = 1-4; y, z = 0-2], were prepared as angiotensin converting enzyme inhibitors (no data). Thus, N-(2-indanyl)glycine phenylmethyl ester (preparation given) and S-PhCH2O2CNH(CH2)4CH[OP(O)(OH)(CH2)4Ph]CO2H were condensed using DCC/Et3N in THF and the product was hydrogenolyzed in MeOH over Pd/C followed by treatment with LiOH to give I dilithium salt.

L6 ANSWER 47 OF 48 EMBASE

ACCESSION NUMBER: 1990289737 EMBASE
TITLE: Antihypertensive medications: Relative effectiveness and adverse reactions.
AUTHOR: Moser, M.
CORPORATE SOURCE: Yale University, School of Medicine, New Haven, CT, United States.
SOURCE: Journal of Hypertension, (1990) Vol. 8, No. SUPPL. 2, pp. S9-S16.
ISSN: 0263-6352 CODEN: JOHYD3
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
006 Internal Medicine
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 13 Dec 1991
Last Updated on STN: 13 Dec 1991

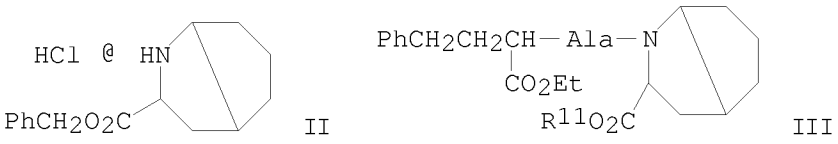
AB Thiazide diuretics may be more effective as antihypertensive agents in many subsets op patients than other medications, especially in reducing systolic blood pressure. Angiotensin converting enzyme (ACE) inhibitors and β-blockers are less effective than calcium blockers or diuretics in black hypertensives and

β-blockers may be less effective in the elderly. Calcium blockers are equally effective in blacks, whites and the elderly but may not be as efficacious as diuretics. When used as initial monotherapy, most available antihypertensive drugs produce significant adverse subjective effects in about 8-10% of patients; centrally acting drugs, however, may produce annoying side effects in 20-30% of patients. Usually, some medication can be found that lowers blood pressure and is acceptable to the patient. Adverse metabolic effects are probably of limited long-term clinical significance except in a few patients. An approach to therapy that will prove effective in a majority of patients is outlined. Any one of the four classes of agents may appropriately be used as initial monotherapy (diuretics, β-blockers, ACE inhibitors , calcium blockers). Diuretics are recommended as the second drug of choice if one of the other agents is used first. With this approach approximately 80% or more of patients can be controlled at normotensive levels on one or at most two drugs.

L6 ANSWER 48 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
ACCESSION NUMBER: 1985:560859 CAPLUS
DOCUMENT NUMBER: 103:160859
ORIGINAL REFERENCE NO.: 103:25849a,25852a
TITLE: N-Alkylated dipeptides and their esters
INVENTOR(S): Teetz, Volker; Wissmann, Hans; Urbach, Hansjoerg
PATENT ASSIGNEE(S): Hoechst A.-G. , Fed. Rep. Ger.
SOURCE: Eur. Pat. Appl., 33 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 135182	A2	19850327	EP 1984-110678	19840907
EP 135182	A3	19860305		
EP 135182	B1	19880727		
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
DE 3333454	A1	19850411	DE 1983-3333454	19830916
AT 35997	T	19880815	AT 1984-110678	19840907
HU 36145	A2	19850828	HU 1984-3415	19840910
HU 201565	B	19901128		
FI 8403590	A	19850317	FI 1984-3590	19840913
FI 80464	B	19900228		
FI 80464	C	19900611		
CA 1338163	C	19960312	CA 1984-463078	19840913
DK 8404405	A	19850317	DK 1984-4405	19840914
DK 164939	B	19920914		
DK 164939	C	19930201		
NO 8403662	A	19850318	NO 1984-3662	19840914
NO 167743	B	19910826		
NO 167743	C	19911204		
AU 8433070	A	19850321	AU 1984-33070	19840914
AU 576782	B2	19880908		
JP 60089497	A	19850520	JP 1984-191868	19840914
JP 07098835	B	19951025		
ZA 8407257	A	19850529	ZA 1984-7257	19840914
IL 72947	A	19890228	IL 1984-72947	19840914
US 5068351	A	19911126	US 1990-560004	19900727
PRIORITY APPLN. INFO.:			DE 1983-3333454 A	19830916
			EP 1984-110678 A	19840907
			US 1984-650715 B1	19840914
			US 1986-943882 B1	19861219

US 1988-178767 B1 19880330
US 1989-403919 B1 19890907
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): MARPAT 103:160859
GI



AB Title compds. R3O2CCHR4NR5COCHR1NHCH(CO2R2)(CH2)nR [n = 1, 2; R = H, (un)substituted C1-8 aliphatic, C3-9 alicyclic, C6-12 aromatic, C7-14 araliph., or C7-14 alicyclic aliphatic residue, OR6, SR6 [R6 = (un)substituted C1-4 aliphatic, C6-12 aromatic, or heteroarom. residue]; R1 = H, (un)usbsituted C1-6 aliphatic, C3-9 alicyclic, C4-13 alicyclic aliphatic, C6-12 aromatic, C7-16 araliph., or heteroarom. residue, amino acid side chain; R2, R3 = H, (un)substituted C1-6 aliphatic, C3-9 alicyclic, C6-12 aromatic, or C7-16 araliph. residue; CHR4NR5 = C5-15 heterocyclic mono-, bi-, or tricyclic ring system] were prepared via the condensation of HO2CCHR1NHCH(CO2R2)(CH2)nR with R3O2CCHR4NHR5 in the presence of phosphinic acid anhydrides R7R8P(O)OP(O)R9R10 (R7, R8, R9, R10 = alkyl or aralkyl). Thus, (S,S,S)-azabicyclo[3.3.0]octane II was condensed with (S)-PhCH2CH2CH(CO2Et)-(S)-Ala-OH by ethylmethylphosphinic acid anhydride in CH2Cl2 containing Et3N to give peptide III (R11 = CH2Rh), which was debenzylated to give III (R1 = H). I inhibit angiotensin-converting enzyme and can be used as antihypertensives (no data).
OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

=> logoff
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:y

(FILE 'HOME' ENTERED AT 22:23:59 ON 05 MAY 2010)

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 22:24:18 ON 05 MAY 2010
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L2 66 SEA FILE=MFE SPE=ON ABB=ON PLU=ON L1 AND (BONE(W) MORPHOGEN OR OSTEOGENIC(W) PROTEIN OR BMP OR OP)
L3 5814 SEA FILE=MFE SPE=ON ABB=ON PLU=ON L1 AND PROTEINURIA
L4 6 SEA FILE=MFE SPE=ON ABB=ON PLU=ON L2 AND PROTEINURIA
L5 6 DUP REM L4 (0 DUPLICATES REMOVED)
L6 48 DUP REM L2 (18 DUPLICATES REMOVED)
DIS IBIB ABS L5 1-6

FILE 'STNGUIDE' ENTERED AT 22:28:10 ON 05 MAY 2010

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 22:30:34 ON 05 MAY 2010
DIS IBIB ABS L6 1-10

FILE 'STNGUIDE' ENTERED AT 22:30:35 ON 05 MAY 2010

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 22:32:19 ON 05 MAY 2010
DIS IBIB ABS L6 11-20

FILE 'STNGUIDE' ENTERED AT 22:32:21 ON 05 MAY 2010

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 22:34:07 ON 05 MAY 2010
DIS IBIB ABS L6 21-30

FILE 'STNGUIDE' ENTERED AT 22:34:09 ON 05 MAY 2010

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 22:41:37 ON 05 MAY 2010
DIS IBIB ABS L6 31-40

FILE 'STNGUIDE' ENTERED AT 22:41:40 ON 05 MAY 2010

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 22:43:20 ON 05 MAY 2010
DIS IBIB ABS L6 41-48

FILE 'STNGUIDE' ENTERED AT 22:43:21 ON 05 MAY 2010

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.14	199.30

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-10.20

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